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SEKELSELANEMIE (SICKLE-CELL ANAEMIA)*

'N STUDIE VAN 'N KLEURLINGFAMILIE (4 PASIËNTE)

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In die Suid-Afrikaanse literatuur word slegs 4 gevalle met sekelselanemie aangemeld.[1-4] Die geval van Segal, Grusin en Cassel[4] wyk egter in verskeie opsigte af van die klassieke beeld van sekelselanemie, nl. met 'n buitengewone hoë hemoglobiengehalte, sonder transfusie, en 'n vergrote milt; dit sou beter inpas by 'n sekelsel-talassemie as by 'n sekelselanemie, soos aangedui deur Vandepitte.[5] Al hierdie gevalle is vanuit die noordelike gebiede van Suid-Afrika gerapporteer, behalwe dié van Berk en Bull[2] wat beskryf is in 'n Indiër-vrou, gebore in Durban. Ons pasiënte is die enigste gevalle (sover beskryf) vanuit die Kaapse Skiereiland, wat ook hier gebore is. Hierdie 2 gevalle is dus die 5e en 6e gevalle wat in die Suid-Afrikaanse literatuur gerapporteer word.

Die pasiënt, 'n 29-jarige Kleurlingvrou, asook haar vader, moeder en enigste suster (sy het geen broers nie) was beskikbaar vir studie. Die vader is van gemengde Ierse, Javaanse en plaaslike Kleurlingbloed (Blank en Bantoe), en sommige van sy grootouers kom van St. Helena-eiland. Die moeder is gebore van Kaapse Kleurlingouers te Paarl, en sy het daar grootgeword. Haar ouers is albei daar gebore en 'n Ierse voorvader was die mees onlangse blanke bloedtoevoeging tot die familie. Bloedgroepbepalings is vir al die lede gedoen om 'n opvallende buite-egtelike kindskap uit te skakel.

Die pasiënt was ook swanger by toelating en dit is die eerste geval van swangerskap by 'n sekelselanemie-lyer wat in die Suid-Afrikaanse literatuur vermeld word. In die wêreldliteratuur het Eisenstein, Posner en Friedman[6] slegs 129 beskrewe gevalle gevind, en 'n verdere 9 bygevoeg. Dit het die totaal in die wêreldliteratuur op 138 te staan gebring.

Geval 1

Die pasiënt is 'n 29-jarige Kleurlingvrou wat op 15 April 1958 toegelaat is tot die Karl Bremer-Hospitaal, met die klagte van 'n aanval van akute stekende pyne in die regter-hipokondrium sedert 11 dae voor opname. Die pyn duur onafgebroke voort en straal nie uit na die skouer, rug of af in die buik nie. Die pyn het geen verband met etes nie.

Twee dae na hierdie pynaanval het sy ook erger pyn in die regter-elmboog en -skouer ontwikkel. Die pyn word nie deur beweging vererger nie, en is aanhoudend. Die knieë is ook effe pynlik maar veel minder as die elmboog en skouer.

Sy het reeds 'n paar maal vantevore sulke aanvalle van gewrigspyne ondervind, en is deur haar private geneesheer vir rumatiekkoors behandel. Haar toestand was ook, as 'n buitepasiënt by 'n ander hospitaal, as rumatiekkoors gediagnoseer en behandel. By hierdie geleentheid is sy meegedeel dat haar hart vergroot is as gevolg van rumatiese klepletsels.

Die geneesheer wat haar na die hospitaal gestuur het, het geelsug in die sklera opgemerk, maar sy is reeds vanaf 5-jarige ouderdom daarvan bewus dat haar oë geel is. Sedert kindsdae is sy geneig tot neusbloedings. Sy het frekwensie (4 tot 5 maal snags) maar geen hematurie nie. Die urine is donkerder tans as voorheen. Sy word gou moeg by inspanning en is baie jare lank al kortasem as sy trappe klim of vinnig loop. Sy kry soms hartkloppings.

Haar laaste normale menstruasie was op 20 November 1957 en dit het 2-3 dae lank geduur. Sy ondervind dismenoree tydens menstruasie. Sy is tans 5 maande swanger. Die vorige swangerskap het op 8 maande geëindig met 'n doodgebore kind. Geen afwyking kon aan die kind bemerk word nie. Die geboorte het tuis geskied.

Haar suster het op 6-jarige ouderdom ook geelsug ontwikkel en haar oë is by tye geel, maar minder opvallend so as dié van die pasiënt (kyk hierna).

Bevindinge by ondersoek. 'n Jong Kleurlingvrou, 22 weke swanger. Die romp kom enigsins kort voor in vergelyking met die ledemate (Afb. 1). Die sklera is duidelik ikteries, en die slymvliese kom anemies voor. Geen trommelstok-vingers of splinter-

Afb. 1. Foto van die pasiënt na behandeling. Romp betreklik kort in vergelyking met ledemate, maar nie opvallend nie. Die gelaatstrekke is meer Asiaties as tipies Kleurling.

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bloedings onder die naels nie. Temperatuur 99°F. Pols 100 per minuut. Bloeddruk 125/70 mm. Hg.

By ondersoek van die retina is daar duidelike en opvallende kronkeling van die retinale vate. (Hierdie kronkeling het kommentaar van alle waarnemers uitgelok, maar kon nie fotografies vasgelê word nie.) Geen ander afwykings is waargeneem nie. Die hart is klinies vergroot met 'n diffuse, effe heffende, soort puntstoot net buite die middel-klavikulêre lyn in die 5e interkostale ruimte. Daar is 'n graad II sistoliese geruis oor die hele prekordium hoorbaar, en die P 2 is verdubbel. 'n Paar basale krepitasies en enkele ronchi oor beide longe is hoorbaar. In die buik is die lewerrand net tasbaar, maar die milt is nie tasbaar nie. Die fundus uteri se grootte dui 'n swangerskap van 20-22 weke aan. Neurologies is die kraniale senuwees normaal, die sensorium is normaal en die reflekse is normaal. By die ledemate is daar drukteerheid oor die mediale end van die clavicula regs, en oor die regter-skouergewrig en veral die regter-elmboog. Daar is geen swelsel, rooiheid of verskil in huidtemperatuur oor hierdie gewrigte nie. Daar is geen seer of trofiese velveranderings nie.

Spesiale ondersoeke op 16 April 1958—Die resultate van bloedondersoeke was soos volg: Hemoglobien 6·5 g.%, witselle 13,000 per c. mm., polimorfe leukosiete 58%, limfosiete 36%, monosiete 3%, eosinofiele 1%, bloedplaatjies normaal en besinking 85 mm. per eerste uur (Westergren).

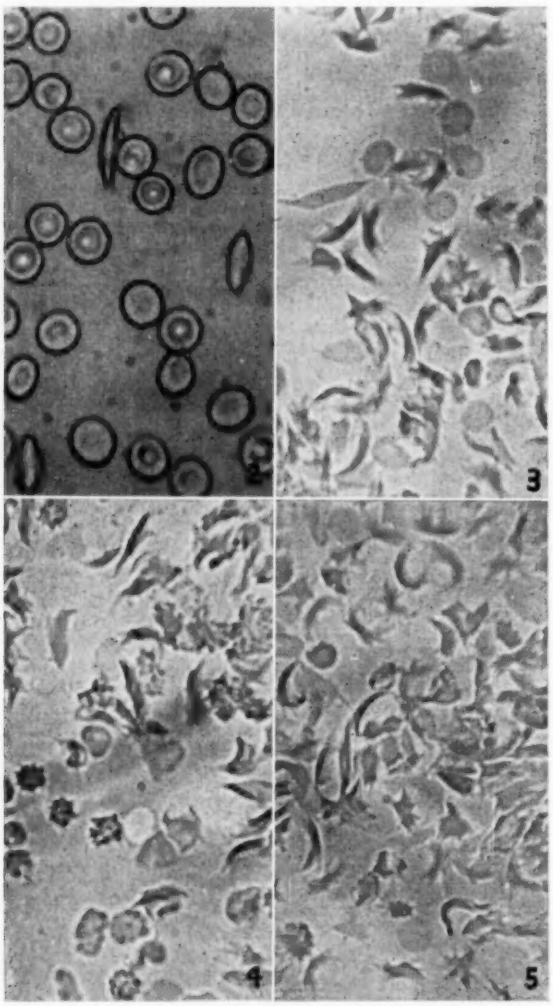
Ondersoek van die urine het die volgende opgelewer: S.G. 1,007, albumien ++, diasynsuur +, bilirubien + (slegs by toelating gevind), urobilinogeen + en 12 rooibloedselle/H.V.V. Die bloed Wassermann—reaksie was negatief.

Lewerfunksie-proewe het die volgende getoon: Albumien 3·5 en globulien 3·2 g.%, timolturbiditeit 1, timolflokkulasie 0, sinksulfaat turbiditeit 4, serumbilirubien 7·2 mg.%, direk en indirekte van den Bergh +, serumcholesterol 262 mg.%, serum alkaliese fosfatase 6·6 eenhede en protrombien-indeks 100+. Die retikulosiettelling was 10%.

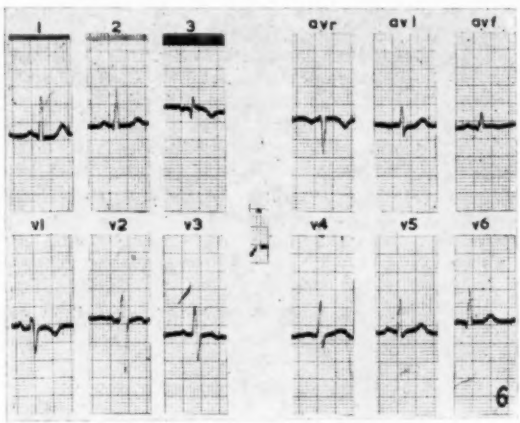
Beenmurg-ondersoek toon 'n aktiewe beenmurg met 'n normale differensiële telling, en 'n paar skyfselle (target cells). Diagnose op hierdie stadium was (1) mitralis insuffisiënsie as gevolg van rumatiese aantasting (en moontlik subakute bakteriële endokarditis) en (2) hemolitiese anemie.

By herondersoek van die beenmurg is selle ontdek wat sekelvormig of hawervormig voorkom (Afb. 2). Die pasiënt het op hierdie stadium reeds 3 pinte gepakte selle ontvang vir anemiese klagtes en hartversaking wat begin intree het. Die bloed is *in vitro* vir die verskynsel van sekeling getoets en dit was positief reeds onmiddellik by ondersoek (±15 min.). Afbs. 3-5 is geneem na 2, 6 en 24 uur respektiewelik. Die toets is gedoen deur 'n bandjie vir 6 minute om die vinger te laat staan en 'n druppel bloed word dan afgeseël onder 'n dekglasie. 'n Reduserende middel is nie bygevoeg nie. Die oorspronklike bloedsmere was nie meer beskikbaar nie, maar op 'n preparaat vir ondersoek na Lupus erythematosus-selle, kort na toelating, is sekelvormige rooibloedselle gevind. Roentgenfotos van die hart toon 'n hart na links en regs vergroot en ook verwyding van die murgholte en areas van beenverdigting in die tibia. Radiologiese ondersoeke is tot 'n minimum beperk vanweë die swangerskap van die pasiënt.

Die elektrokardiogram toon moontlike regter ventrikulêre spanning, maar is origens binne normale perke (Afb. 6). PQ-tyd 0·16 sekondes.



Afb. 2. Beenmurg van die pasiënt waarop sekelvormige selle eerste ontdek is. Daar is ook 'n paar skyfselle (target cells) op die smeer.
Afb. 3. Sekelselle is *in vitro* gedemonstreer binne enkele minute na die bloed verkry is. Die foto toon die mate van sekeling na 2 uur. Ongekleurde preparaat. (Bloed van die pasiënt).
Afb. 4. Sekeling *in vitro* na 6 uur. Bloed van die pasiënt.
Afb. 5. Sekeling *in vitro* na 24 uur. Bloed van die pasiënt.
Die normale selle nog sigbaar, is waarskynlik te wyte aan die bloedtransfusies.



Afb. 6. Elektrokardiogram toon moontlike R.V. spanning, origens binne normale perke. (PQ=0·16 sekondes.)

Verdere ondersoeke. 'n Differensiële besinking is gedoen op veneuse en deeglik geoksigeneerde bloed. Die verskil in besinking is slegs positief in gevalle met sekelselanemie.[7] Die resultate word hieronder getabuleer (Westergren se metode).

Geval 2

Kleurlingvrou, 25 jaar oud—suster van die pasiënt. Sy is tans volkome asimptomaties. Sy het ‚jigpyn'-aanvalle gehad en is

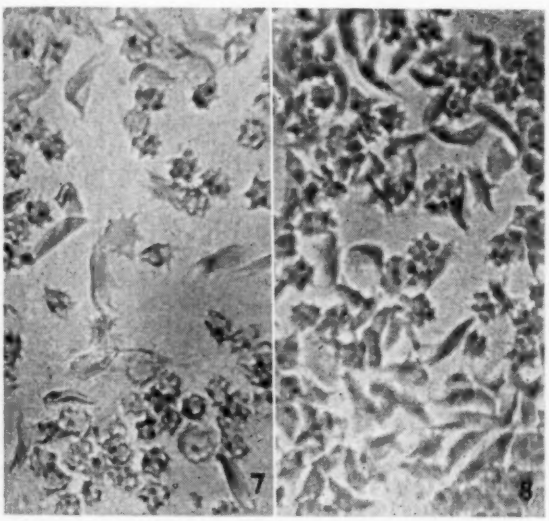
TABEL I. DIFFERENSIËLE BESINKING

	Geoksigeneerde bloed	*Veneuse bloed*
½ uur	35 mm.	12 mm.
1 uur	62 mm.	25 mm.
1½ uur	84 mm.	44 mm.
2 uur	94 mm.	54 mm.

Die bloedgroep van die pasiënt is O cDe/cde NN.

ongeveer 18 maande gelede vir subakute bakteriële endokarditis in 'n naburige hospitaal behandel. Sy het geelsug gehad op die ouderdom van 6 jaar en is sedertdien voortdurend ‚gelerig in haar oë', by tye is dit erger en by tye beter.

Bevindinge by ondersoek. Die sklera kom nie geel voor nie, maar slymvliese lyk effe anemies. Daar is 'n sagte sistoliese geruis, graad I, oor die hele prekordium. Die hart is nie klinies vergroot nie. Daar is kronkeling van vate in die oogvelde dog minder duidelik as in die geval van haar suster. Die lewer en milt nie tasbaar nie. By radiologiese ondersoek is die skedel normaal, die hart net binne normale perke en die langbene normaal. Elektrokardiografiese bevinding is binne normale perke. PQ-tyd is 0·20 sekondes. Die resultate van bloedondersoek was soos volg: Hemoglobien 9·5 g.% en witselle 11,750 per c. mm. Die perifêre smeer bevat sekelselle. Die *in vitro* toets vir sekeling was positief (Afbs. 7 en 8). Differensiële besinking (Westergren-metode) is soos volg:



Afb. 7. *In vitro* sekeling van die pasiënt se suster se rooibloedselle (ongekleurde preparaat). Sekeling na 12 uur. Byna al die selle is misvormd.

Afb. 8. *In vitro* sekeling na 24 uur. Al die rooibloedselle is in die sekelvorm.

TABEL II. DIFFERENSIËLE BESINKING

	Geoksigeneerde bloed	*Veneuse bloed*
½ uur	32	3
1 uur	80	5
1½ uur	122	6
2 uur	128	6

Die bloedgroep van die pasiënt is B cde/cde NN Kell—Fy^a+.

Geval 3

'n Kleurlingman, ±55 jaar oud, vader van die gesin.

Hy is in 'n inrigting vir kroniese siekes as gevolg van 'n hemiplegie en disartrie, te wyte aan 'n serebrovaskulêre ongeluk. Hy het geen klagtes nie en bevindings by ondersoek, behalwe vir die hemiplegie, is negatief. Daar was nooit hematurie nie.

Die resultate van bloedondersoek was soos volg: Hemoglobien 14·6 g.% en witselle 7,800 per c. mm. Sekeling *in vitro* was langsaam en minder duidelik as dié van sy dogters na 24 uur. *Sy bloedgroep* is O $CD^{u}e$/cde NN.

Geval 4

'n Kleurlingvrou, ±45 jaar oud, moeder van die gesin. Daar is tekens van vetsug, maar origens is sy asimptomaties. Daar was nooit hematurie nie. Daar is osteoartritis van die hande, maar verder geen afwykings nie.

Die resultate van bloedondersoek was soos volg: hemoglobien 14·3 g.% en witselle 7,000 per c. mm. Sekeling *in vitro* is soos die vader s'n. Haar *bloedgroep* is B cde/cde NN.

Elektroforese

Die elektroforese is gedoen by pH 6·8. Gewoonlik word dit by pH 8·6 of 8·8 gedoen. Die beweeglikheid word geaffekteer namate die elektroforese gedoen word verder of nader aan die iso-elektriese punt van die proteïen. Hoewel by pH 8·6 die meeste abnormale hemoglobienes stadiger beweeg as normale hemoglobien, is dit nie noodwendig die geval by 'n laer pH nie. Sekelsel-hemoglobien was ongelukkig nie beskikbaar vir vergelyking nie. Die elektroforetiese diagram dui egter daarop dat die bloed van die pasiënt, die vader en die moeder, min of meer dieselfde samestelling het. Ons weet dat hulle almal *in vitro* sekel, dus is daar sekelsel-hemoglobien aanwesig. Ons weet ook dat die pasiënt hemoglobien A transfusies ontvang het, en dus is haar beeld Hb·AS en so ook dié van die ouers. Die elektroforetiese waardes is soos volg: Normaal 4·9, moeder 5·4, vader 5·2, pasiënt 5·3, suster 5·8 en normaal 4·6.

BESPREKING

Die Kell- en Duffy-groepe kon slegs vir die suster van die pasiënt gedoen word, waarna die serum opgeraak het. Die ABO groepe dui daarop dat die moeder genotipies BO moet wees, aangesien die dogters B en O is. Hiervolgens, en volgens die cde en MNS groepe, kan die kinders binneegtelik wees, maar dit dien natuurlik nie as bewys daarvan nie. Altwee ouers toon sekeling, maar is asimptomaties. Hulle is dus heterosigoties vir die sekelselgene. Indien 'n ander abnormale hemoglobien teenwoordig was, bv. C, D of talassemie, sou hulle nie asimptomaties gewees het nie.

Die elektroforetiese beeld dui daarop dat die hele familie 'n abnormale hemoglobien besit. Die suster het geen bloedtransfusies ontvang nie en aangesien sy anemies is, (Hb. 9·5 g.) is sy homosigoties vir hierdie hemoglobien. Die pasiënt, as gevolg van haar bloedtransfusies, toon dus 'n heterosigotiese patroon, te wyte aan normale hemoglobien in die getransfuseerde bloed.[8] Almal toon sekeling *in vitro*, maar sekelselle kon slegs in die pasiënt en haar suster se perifêre bloed gevind word. Die abnormale hemoglobien is dus hemoglobien S. Die ouers het dus die sekelseleienskap en albei hulle dogters het sekelselanemie.

Hoewel die sekelseleienskap nagegaan is in die Kaapse Kleurling[9] (voorkoms 0·58%), is sekelselanemie nog nie vanuit die Kaapprovinsie beskryf in 'n plaaslik gebore Kleurling nie. Die geval van Berk en Bull[2] is vanuit die Kaapprovinsie beskryf in 'n Indiërvrou gebore in Durban. Die verhouding van die voorkoms van die eienskap tot die anemie wissel van streek tot streek, en die verhouding in Afrika word op 1,000 : 1 gestel.[10] Die voorkoms in die Kaapprovinsie moet dus as 0·006% gestel word en die toestand is dus uiters seldsaam. As gevolg van die geografiese saamgroepering van die Kleurlingbevolking en hulle hoë geboortesyfer, mag die toestand mettertyd meer volop word.

Die probleme in verband met differensiële diagnose is

bekend en die dikwelse verwarring met subakute bakteriële endokarditis en rumatiekkoors word weereens in hierdie gevalle geïllustreer.

'n Osmotiese fragiliteitstoets is vir die pasiënt gedoen voor die diagnose van sekelselanemie gemaak is, maar daar is nie toegelaat vir die feit dat sy ikteries (bilirubien 5 mg.%) was nie, en hemolise is gerapporteer vanaf die eerste buis vanweë die geel kleur. Die ondersoek is nie later herhaal nie, vanweë die bloedtransfusies wat sy ontvang het. Die suster kon nie weer kom vir verdere ondersoek nie.

Die pasiënt se swangerskap word tans verder deur die verloskundige departement van hierdie hospitaal gehanteer. Die literatuur aangaande swangerskap en sekelselanemie is noodwendig beperk.[6] 'n Kompromis moet getref word tussen die anemie wat die vrug nadelig beïnvloed en bloedtransfusies wat, indien die hemoglobien te hoog sou gaan, neig om beenmurg-aktiwiteit te onderdruk.

Die prognose van sekelselanemie is nie met sekerheid bepaalbaar in gevalle met swangerskap nie, maar Eisenstein *et al.*[6] vind dat 30 uit die 138 beskrewe gevalle tydens swangerskap oorlede is. Toksemie van swangerskap kom effens meer dikwels voor in sekelselanemie as in die gemiddelde bevolking. Kraam as sodanig word nie geaffekteer nie. Volgens hierdie skrywers[6] moet vitamine, yster en lewerekstrak in die pre-natale periode gegee word. Bloedtransfusie is, volgens hulle, nie aangedui tensy die hemoglobien onder 8 g.% daal nie. Infeksies, waartoe hierdie gevalle baie geneig is, vereis hospitalisasie. Die fetale verlies in gevalle met sekelselanemie en swangerskap, is 41·9%. Die hoë moederlike sterftesyfer regverdig sterilisasie volgens genoemde skrywers.

Die gevaar om die hemoglobien van hierdie pasiënte bo 10 g. te bring, lê in die feit dat 'n aplastiese of aregeneratiewe krisis kan ontstaan. Dit is ook die rede waarom suurstof nie vir te lang periodes aaneen gegee moet word nie. Die albuminurie, roobloedselle in die urine en 'n S.G. wat tussen 1,007-1,010 varieer, moet waarskynlik as 'n aanduiding van vroeë renale skade gesien word, aangesien dit algemeen is en gewoonlik die oorsaak van dood in die derde dekade is. By hierdie pasiënt was die moeder se mense ten minste twee geslagte lank plaaslik woonagtig, terwyl die vader se grootouers op St. Helena-eiland gebore is. Meer besonderhede as dat van sy voorsate van Javaanse afkoms is, kon nie verkry word nie.

OPSOMMING

1. 'n Studie van 'n Kleurlingfamilie met die sekelseleienskap in beide ouers, en sekelselanemie in albei die kinders, word beskryf.

2. Ons meen dat dit die eerste beskrywing is in die Suid-Afrikaanse literatuur van sekelselanemie in 'n pasiënt wat in die Kaapse Skiereiland gebore is. Vier gevalle is reeds beskrywe,[1-4] almal vanuit die noordelike dele van Suid-Afrika, behalwe dié van Berk en Bull, wat in Durban gebore is, maar vanuit die Kaapprovinsie beskrywe is.

3. Bloedgroep-studies toon geen bewys van buite-egtelike kindskap nie.

4. Dit is die eerste geval van swangerskap by 'n sekelselanemie-lyer wat in die Suid-Afrikaanse literatuur beskrywe is. Die wêreld-literatuur bevat slegs 139 gevalle van swangerskap by sekelselanemie.[6]

5. Elektroforetiese bevindings word bespreek. Die besinkingstoets van Winsor en Burch[7] is positief in die twee anemiese lede van die familie.

6. Die kliniese beeld in hierdie gevalle is tipies behalwe vir hulle ouerdom; dit sluit egter nie die diagnose uit nie.

SUMMARY

1. A study was made of a Cape Coloured family showing *in vitro* sickling in the father and mother, both being asymptomatic with a normal haemoglobin and white blood cell count. Sickle-cell anaemia was discovered in our patient and in her sister, who has previously been treated elsewhere for sub-acute bacterial endocarditis.

2. It is believed to be the first reported case of sickle-cell anaemia born in the Cape Province. Four other cases have been reported[1-4] in the South African literature, one of which[4] might have been sickle-cell thalassaemia because of some aberrant findings as pointed out by Vandepitte.[6] The case of Berk and Bull[2] was born in Durban but was reported from Cape Town.

3. Blood-group studies show no evidence of false parentage, but do not prove the daughters to be the offspring of their father.

4. The sedimentation-rate test of Winsor and Burch (on aerated and venous blood) is positive in the two anaemic members of the family. It is said to be positive only in cases of sickle-cell anaemia.[7]

5. The clinical picture in our cases is completely consistent with the typically described picture, except for their ages which, though rare, do not exclude the diagnosis.

6. Electrophoreses was done at pH 6·8 and shows an abnormal haemoglobin in all members of the family. The electrophoretic pattern of the patient's haemoglobin is approximately the same as that of the parents' while the sister's, who received no blood transfusions, is faster than that of the rest of the family. The parents, being asymptomatic, are therefore heterozygotic for the abnormal haemoglobin; the patient's electrophoretic pattern being made so by transfusions. As sickling is present in all members, the abnormal haemoglobin is HbS. No known HbS is available for control. Electrophoreses on abnormal haemoglobin is usually done at pH 8·6 or 8·8. This lower pH (6·8) effects the mobility of the haemoglobin.

7. Pregnancy (with sickle-cell anaemia) has been reported in 139 cases in the world literature.[6] Our patient, being pregnant, gave rise to some problems in the management of her pregnancy and anaemia. It is the first case of pregnancy with sickle-cell anaemia reported in the South African literature.

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VERWYSINGS

1. Altman, A. (1945): Clin. Proc., **4**, 1.
2. Berk, C. en Bull, G. M. (1943): *Ibid.*, **2**, 147.
3. Grek, I. J. en Findlay, M. (1951): S. Afr. T. Geneesk., **25**, 780.
4. Segal, F., Grusin, H. en Cassel, R. (1956): *Ibid.*, **30**, 63.
5. Vandepitte, J. (1956): *Ibid.*, **30**, 392.
6. Eisenstein, M. I., Posner, A. C. en Friedman, S. (1956): Amer. J. Obstet. Gynec., **72**, 622.
7. Winsor, T. en Burch, G. E. (1944): Amer. J. Med. Sci., **207**, 152.
8. Nadel, J. A. en Spivack, A. P. (1958): Ann. Intern Med., **48**, 399.
9. Esrachowitz, S. R., Friedlander, S., Radloff, G. en Saunders, S. (1952): S. Afr. T. Geneesk., **26**, 239.
10. Wintrobe, M. M. (1956): *Clinical Hematology*, 4e ed. Londen: Henry Kimpton.

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EDITORIAL : VAN DIE REDAKSIE

A DRINKING PROBLEM

It is not hard to see why doctors have been so slow to accept the alcoholic as a sick man. For, in fact, alcoholism is only in part an illness. Firstly, the condition develops on the basis of personal habits and, secondly, treatment is only possible when the alcoholic agrees to accept it and himself work for it. In a sense, one chooses to be an alcoholic and has to choose to stop being one. The doctor, after all, a product of his culture, may find himself disapproving morally of his alcoholic patient, prepared to treat what illness there is but unable to overlook the 'lack of will power'.

The doctor may also become impatient at having to wait for his patient to concede that he is ill and in need of treatment. A man dangerously diseased from drink, his life in ruins about him, often will hold obstinately that he is not an alcoholic. (He can stop if he wants to—only he has not wanted to yet. And why shouldn't he be able to drink when other people can?) Alcoholics Anonymous has a tragic slogan that a man cannot be helped until he has hit rock bottom. However, it would seem that the doctor, through his calling, has a powerful weapon which can save many drinkers before they are physically or socially decrepit: if the alcoholic is permitted to say that he suffers from an illness, he thereby obtains the privilege and the right to seek a doctor to treat his illness. The misfortune, of course, remains that he may now consult a doctor too prejudiced to undertake to treat him, or so uninformed about alcoholism that he permits the alcoholic's touchy aggressiveness to antagonize him.

That alcoholism has become regarded as an illness is therefore a step of tremendous consequence to the alcoholic. He need not cringe as a weakling, or guiltily come forth as a sinner. He has the status of a patient. In 1956 the American Medical Association had unanimously passed by its House of Delegates a resolution that alcoholics are valid patients and should be treated as such. This was another step forward, as great as had happened in 1939 when an alcoholic layman searched for an alcoholic physician in Akron, Ohio, and together they founded Alcoholics Anonymous.

In accord with this enlightened attitude is the publication,[1] in the Modern Monographs in Industrial Medicine series, of a handbook for physicians about the management of alcoholic patients. Most doctors get confronted by some aspect of alcoholism in their work, either when treating the families of drinkers or the drinkers themselves.

Psychological factors are central in the causation of alcoholism. Therefore, after the patient has been 'dried out', he needs medical aftercare while he attempts to regain a place in the community. He may often need psychiatric guidance in understanding his inability to tolerate stress. As he learns new techniques for handling the anxiety roused in him by work or social demands, Alcoholics Anonymous may afford him most substantial support. However, many ex-drinkers cannot tolerate the confessional and spiritual orientation of this organization, and because of this and for treatment of deeper character distortion, they will need medical psychotherapy.

An important medical aid, if correctly advised, is Antabus, which the drinker can use as a crutch once on the drug, which he should take each morning so regularly that it becomes a habit. He knows he will not enjoy a drink unless he gives the drug three days to leave his system. He thus provides himself with a period of grace, during which he can rethink whether it is necessary for him to start up on the drink again. Often enough, in this time, he deals with the problem facing him, and recommences his Antabus and not his drinking.

It may be that a doctor working regularly with alcoholics, gets to know them best and assists them most easily. Nevertheless, the profession as a whole should be aware of the responsibility to the alcoholic which contemporary medicine admits, and of the valuable human material that can be saved, and family life protected, through an intelligent approach to the men and women who have a drinking problem.

1. Pfeffer, A. Z. (1958): *Alcoholism*. New York and London: Grune and Stratton.

AKADEMIESE JAARDAG—MEDIESE SKOOL STELLENBOSCH

Dit is 'n ou en bekende gebruik by akademiese inrigtings om op gesette tye spesiale byeenkomste te reël wat die geleentheid bied om die ontstaan van die inrigting in feestelike herinnering te roep; wat dien as vertoonvenster van die kulturele bedrywighede van die betrokke inrigting, maar wat eintlik, en veral, 'n geleentheid bied om die doel en strewe en voortbestaan van die inrigting in kritiese voorbetragting te neem.

Hierdie gebruik is nodig en goed en gesond. So 'n gereelde byeenkoms kan, as dit reg aangevoor en uitgebou word, een van die belangrike hoekstene word waarom 'n wyere akademiese tradisie groei en gedy. Dit kan lei tot intellektuele selfondersoek van die lede van die inrigting, tot bekendstelling van sy doel en strewe en metodes aan 'n wyere wêreld en tot die aanblaas van 'n vonk van wetenskaplike en humanitêre entoesiasme wat, veral in die geval van 'n moderne geneeskundige fakulteit, onontbeerlik is.

Met die viering van die tweede akademiese jaardag van die mediese skool van die Universiteit van Stellenbosch en die Karl Bremer-Hospitaal, Bellville, is dit duidelik dat so 'n tradisie besig is om daar te ontwikkel. Soos dit die geval met die eerste jaardag was, het die byeenkoms die vorm aangeneem van 'n feesdag met 'n program wat bestaan het uit wetenskaplike referate en uitstallings, afgewissel deur gesellige sosiale verkeer.

Elders in ons blad (in die rubriek *In die Verbygaan*)

publiseer ons meer besonderhede aangaande die verrigtinge van die program van die jaardag soos dit afgehandel is. Hier wil ons hoofsaaklik die beginsel van dié soort byeenkoms vermeld en aanprys, en ons wil dit in die vooruitsig stel dat hierdie soort akademiese byeenkoms 'n vaste beslag sal kry aan die mediese skool waar dit ontstaan het; ook dat soortgelyke of ooreenkomstige gebruike by ons ander mediese skole sal ontwikkel.

Geleenthede soos hierdie sou dan in die toekoms benuttig kon word, nie slegs om 'n akademiese program van die betrokke inrigting self uit te voer nie, maar ook om kritiese en welwillende verteenwoordigers van al die ander suster-universiteite bymekaar te bring met die doel om vriendskaplike betrekkinge aan te knoop en te bestendig en om saam te beraadslaag oor die groot en belangrike verantwoordelikhede wat daar rus op 'n moderne geneeskundige skool en die produkte wat hy lewer.

‚THE HISTORY OF MEDICINE IN SOUTH AFRICA'

Die eerste besending van die bogenoemde boek, geskryf deur dr. Edmund Burrows, is gereed vir verspreiding. Lede sal onthou dat die Mediese Vereniging onderneem het om hierdie boek uit te gee as 'n diens aan sy lede en aan die land.

Alle lede van die Mediese Vereniging van Suid-Afrika is geregtig op een eksemplaar van die boek, gebind in luukse band en teen die voorkeurprys van 25s. (Die boek, wat in 'n linneband gebind is, sal aan die algemene publiek beskikbaar wees teen 63s.) Met die goedkeuring van die Mediese Vereniging (en in ooreenstemming met die gebruik van Verenigings wat boeke van besondere belang publiseer om aan hulle lede die geleentheid te gee om voorkeur te geniet by die verkry van eksemplare), sal die uitgewer een eksemplaar aan elke lid stuur tensy hy alreeds die aanbod afgewys het. Ook sal elke lid die reg hê om die boek na insae, en binne 'n beperkte tyd, terug te stuur; ons hoop nie dat dit sal gebeur nie.

Die boek is die eerste uitvoerige verslag van die geskiedkundige ontwikkeling van die mediese praktyk in Suid-Afrika en dit gaan die interessante geskiedenis, van die begin af, na van die stryd teen siekte en ongesteldheid in hierdie land tot die einde van die 19e eeu.

Hierdie boek wat uitgegee is onder beskerming van die Mediese Vereniging van Suid-Afrika, kan beskou word as die amptelike geskiedenis van die medisyne in Suid-Afrika. Die Vereniging het volle vertroue dat elke lid graag hierdie waardevolle stukkie Africana sal wil besit.

CONGENITAL TRACHEO-OESOPHAGEAL FISTULA*

A REVIEW ILLUSTRATED BY 34 CASES FROM THE CHILDREN'S HOSPITAL, JOHANNESBURG

DAVID ADLER, F.R.C.S. (EDIN.), *Thoracic Surgeon, Johannesburg*

Congenital tracheo-oesophageal fistula, which is not uncommon, is inevitably fatal without treatment and the diagnosis cannot be delayed for any length of time after birth if success is to crown any surgical endeavour. The results, however, considering the magnitude of the anatomical problem, are encouraging.

PATHOLOGY

In this condition, the proximal cervical segment of the oesophagus ends blindly in a thick-walled hypertrophied pouch, which varies in length from 3 to 4 cm. The upper end of the lower oesophageal segment is usually attenuated and opens by a small fistula 2-6 mm. in diameter into the trachea, less commonly into the carina, and rarely into the right main bronchus. There is always a gap between the distal end of the upper pouch and the proximal end of the distal oesophageal segment at its fistulous opening. Occasionally the two portions of the oesophagus are in close proximity, in others there is a gap of 1 or 2 cm., occasionally up to an inch, and in rare instances, when the fistula opens into the bronchus, a greater gap. Rarely, there is no fistula and the lower end of the oesophagus is atretic and not developed.

Pathological Classification

According to Vogt's classification of atresia (Fig. 1) of the oesophagus there are three groups:[1] viz. (1) Complete atresia, (2) atresia with blind upper and lower pouches, and (3) atresia of the oesophagus with tracheo-oesophageal fistula (*a*) between trachea and upper segment, (*b*) between trachea and lower segment, or (*c*) between trachea and both segments. A 4th group, viz. tracheo-oesophageal fistula without atresia, should be added. Attempted closure of this isolated anomaly was first described by Imperatori[2] in 1939.

Of the published cases 90% fall into Vogt's group 3*a*. Of our 34 cases all but one, which fell into group 2, belonged to group 3a, though the gap between the segments varied considerably.

Embryology

Essentially, according to Saunders,[3] the subdivision of the foregut into its tracheal and oesophageal components is due to ingrowth of two laterally placed septa into the developing lung bud. The failure of complete fusion results in tracheo-oesophageal fistula, whereas undue obliquity of union of these septa results in atresia.

Associated Congenital Anomalies

In 13 of our operated cases associated congenital anomalies were noted; in 3 of them multiple anomalies were present. In only 2 cases were these anomalies incompatible with life. One mongol weighing 7 lb was not operated upon. These anomalies were as follows:

(*a*) *Skeletal Anomalies.* Gross spina bifida 1 case, cervical ribs 4 cases, accessory ribs 3 cases, absent rib 1 case, bifid vertebrae 2 cases, hemivertebrae 1 case (with 3 such vertebrae),

* A paper presented at the South African Medical Congress, Durban, September 1957.

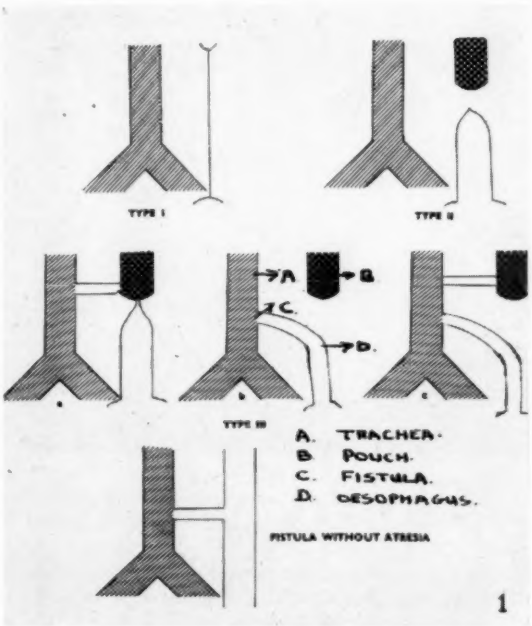

Fig. 1. Congenital tracheo-oesophageal fistula. Diagrams illustrating Vogt's classification.

fused vertebrae 1 case, and 1 case each of bifid right thumb, accessory digits, absent thumb metacarpal, and absent radius.

(*b*) *Cardio-vascular Anaomalies.* One case each of ventricular septal defect, patent ductus, and right aortic arch. One case of tetralogy of Fallot successfully underwent surgery (Fig. 7).

(*c*) *Central-nervous-system Anomalies.* One case each of myelocele, hydrocephalus, and mental retardation.

(*d*) *Gastro-intestinal Anomalies.* Two cases of imperforate anus and 1 case of perineal fistula.

(*e*) *Genito-Urinary Anomalies.* 1 case each of unilateral kidney, horse-shoe kidney, and hypospadias.

(*f*) *Cutaneous Anomalies.* One case with maldeveloped pinnae and imperforate external auditory meati.

HISTORY

Despite the fact that, according to Franklin,[4] the first case was observed by Durston as long ago as 1670 and that the condition was first described by Gibson[5] in 1697, the complete failure of attempts at surgery limited interest in this condition until 1939, when the first surgical success was reported.

Until 1936 all attempts to save these infants were limited to cervical oesophagostomy to allow the upper pouch to drain and gastrostomy to allow for feeding; and in the rare cases which survived an attempt was made to reconstitute an ante-thoracic oesophagus. No case survived these multiple procedures until 1939, when Ladd[6] reported his first survivor in an extensive experience. In Lanman's review[7] (1940) all the 30 cases terminated fatally. By 1940, however, Leven[8] had performed 28 of these multiple-stage operations and 9 patients had lived.

As long ago as 1913[9] Richter had suggested that the ideal surgical treatment would be a direct operation aimed at ligating the fistula and anastomosing the oesophagus end-to-end by a transpleural approach. However, the first direct operation was performed extrapleurally in 1936 by Lanman.[7] Robert Shaw[10] reported the first case to survive the direct operation in 1939. This patient only lived for 12 days and autopsy demonstrated disruption of the anastomosis; Shaw stated that 'leakage from the anastomosis would inevitably occur'. In 1943 Haight and Townsley[11] reported the first survivor from this extrapleural operation, and ushered in a new era of hope in the treatment of these otherwise doomed infants. The first successful case outside the United States was operated upon by Franklin[4] of London in January 1947. In October 1947 Lee MacGregor[12] performed this extrapleural operation in South Africa under local analgesia but the child did not survive more than a few hours. Our first operation was in February 1949 and our first survivor was our third case, in May 1949. These cases were reported by F. W. Roberts.[13] Our first survivor is now 8 years old and has continued to thrive.

In October 1941 Franklin[14] had expressed the view that the extrapleural approach was too tedious, caused too much paradoxical post-operative chest movement, and was so frequently accompanied by minute tears of the pleura that he advised transpleural thoracotomy. He attempted this in 6 cases without success, and he employed the extrapleural approach successfully. Singleton and Knight[15] reported a 7-months survivor after a transpleural repair. Belsey,[16] of Bristol, employed this approach regularly; his first survivor was operated upon in December 1948. Since 1953 we have always used this approach.

INCIDENCE

Before the advent of surgery most infants died of this condition, under the diagnosis of neonatal atelectasis or bronchopneumonia. Haight[17] states that the anomaly occurs once in 2,196 births. Franklin,[18] from the Postgraduate School in London, gave the incidence as 1 in 2,635 deliveries, but Belsey[19] states that the condition is much commoner, occurring once in 800 births. As awareness of the condition amongst doctors spreads so the incidence rises. One of our cases was diagnosed by a midwife 250 miles from Johannesburg. She had attended a lecture at the Queen Victoria Hospital by Dr. Seymour Heymann, who had stressed the presenting symptoms to the class.

Sex Incidence

This anomaly is commoner in males. Of the 31 cases in which the sex was recorded, 21 were males, i.e. 68% (the sex was not recorded of 3 patients who died shortly after operation).

DIAGNOSIS

All neonates have an excess of oral mucus, but these infants with this condition characteristically froth excessively at the mouth; this recurs after aspiration. They have repeated attacks of cyanosis due to overflow and inhalation of saliva and mucus from the proximal pouch. They are avid feeders but immediately return everything unchanged, and the symptoms are aggravated when fluids are given. The abdomen rapidly distends as air passes through the fistula into the stomach *via* the lower oesophageal segment. After this initial phase secondary changes due to inhalation pneumonitis occur. The child is now more constantly cyanosed, with

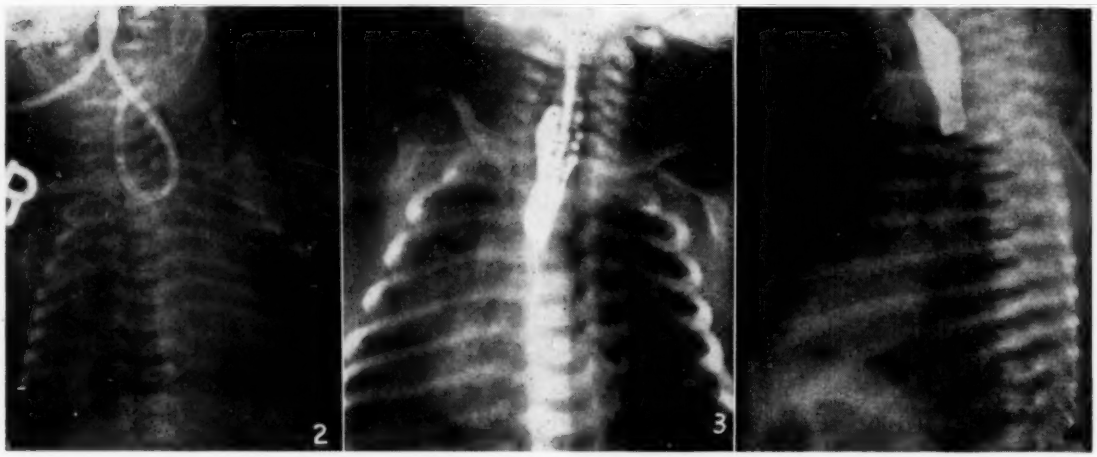

Fig. 2. Radiograph demonstrating coiling of catheter in upper blind pouch.
Fig. 3. Radiograph showing segmental atelectasis of right upper lobe and lipiodol outlining long upper blind pouch.
Fig. 4. Lateral radiograph showing upper pouch ending blindly. Also note air in abdomen indicating fistula between trachea and lower segment.

further exacerbations and progressive dyspnoea. Dehydration is soon followed by inanition and survival for longer than 5-7 days is exceptional—death resulting rather from pulmonary complications than from starvation.

The diagnosis is readily confirmed by the fact that a soft catheter cannot be passed down the oesophagus further than 10 cm. from the gum margin (Fig. 2).

Radiological investigation is useful, not only to confirm the diagnosis but to establish the type of anomaly. Straight films will demonstrate:

(*a*) Excess air in the stomach, indicating the presence of a fistula between the trachea and lower pouch. In such cases it is usually possible to effect an anastomosis.

(*b*) Absence of air in the stomach is commonly associated with an atretic lower segment and the usual anastomosis would be impossible. We have had one such case.

(*c*) The lateral film shows anterior displacement of the trachea with some compression of its posterior margin by the dilated hypertrophied pouch.

(*d*) The presence of inhalation atelectasis or pneumonitis—usually in the right upper lobe. This was present in at least 10 of our cases.

(*e*) The presence of rib and vertibral anomalies is noted. These were present in 8 of our cases.

A lipiodol swallow confirms the diagnosis. This is performed by passing a catheter into the proximal pouch, where it is obstructed at about 10 cm. (Fig. 2) and injecting 0·5 c.c. of lipiodol. Lipiodol outlines the pouch (Figs. 3 and 4) and the rare communication between it and the trachea will be demonstrated. It is essential to aspirate as much of the lipiodol as possible. In case 10 an excess of lipiodol overflowed into the lungs (Fig. 6).

The use of barium sulphate for this purpose cannot be condemned too strongly. The barium causes gross chemical bronchitis; 2 cases (operated on) died 6 and 8 hours after its administration, and one other case had such gross bronchitis that surgery was impracticable.

PRE-OPERATIVE TREATMENT

The *pre-operative treatment* we adopt includes the following points:

1. *Postural drainage*. We used to adopt the Trendelenberg position to lessen spill-over of mucus from the blind upper pouch causing overflow inhalation pneumonitis; but more recently we have nursed the infants slightly head-up to prevent gastric secretions from passing up through the fistula and flooding the tracheo-bronchial tree.

2. Careful repeated intermittent aspiration of pharyngeal mucus and tracheo-bronchial secretions is employed.

3. An ogygen incubator is essential.

4. Steam is used in the incubator and, more recently, despite the suspected risks, alevire has been administered as a fine nebulized spray.

5. Antibiotic therapy is given to control pulmonary infection, viz, 50,000 units of penicillin G and 50 m.g. of streptomycin, both 6-hourly.

Fig. 5. Photograph showing incision used for the extrapleural approach 10 days after surgery.

6. Ascorbic acid is given by injection to stimulate fibroblastic healing.

7. Vitamin K is administered, but not now in such large amounts as in the earlier cases.

8. Pre-operative intravenous hydration is rarely necessary in the first 4 days of life, but a cut-down in the left ankle is always set up immediately before operation for transfusion during surgery and for the assistance of the anaesthetist.

9. Bronchoscopy is performed on the table if there is lobar or pneumonic atelectasis or if there is an excess of tracheobronchial secretion.

Time of operation. We lost case 9, which was operated upon within 24 hours of birth. We feel that operation should not be undertaken till the second day of life, to ensure that postnatal lung expansion is complete. Although in our first 10 cases we operated within a few hours of diagnosis we feel that anaesthetist, theatre staff and surgeon are not able to give of their best in such prolonged and intricate surgery in the early hours; we have therefore prepared our more recent cases for surgery in the forenoon with, I think, improvement in our results.

Pre-operative Complications

1. Prematurity. There were no survivors in the 4 cases weighing less than 5 lb.

2. Atelectasis was present in 10 cases and was responsible for a number of post-operative chest complications.

3. Barium swallow was responsible for the death of 3 infants—2 of whom we unwisely operated upon in the hope of controlling the resultant chemical bronchitis.

4. Lipiodol pneumonitis occurred in one case. This should not happen if only 0·5 c.c. of lipiodol is used and if it is aspirated immediately after X-ray.

5. Ligation of the lower end of the oesophagus was performed in one case to prevent gastric reflux. A feeding gastrostomy was also performed. This infant was referred for surgery on the 27th day of life but unfortunately developed an empyema from leakage at the anastomosis.

6. Neonatal haemorrhage with a large ecchymosis of the abdominal wall was present in case 32. This infant did well after surgery, to die of intracranial haemorrhage on the third day.

THE OPERATION

Anaesthesia

There is no standard anaesthetic for these cases, but adequate ventilation and oxygenation, for which endotracheal intubation is essential, and controlled respiration at the time of anastomosis (because diaphragmatic movement tends to tear out the sutures) are essential for satisfactory transpleural surgery.

If pre-operative bronchoscopy is necessary I prefer doing this under anaesthesia. Diathermy coagulation of bleeding points saves a little time in the exposure but precludes the use of explosive anaesthetics, which may be an undesirable sacrifice for this economy. Once the pleura is entered mobilization is easier with controlled respiration, but it is not essential since the upper lobe is readily controlled with a small retractor. For adequate suturing of the anastomosis, however, *absolute control* of the respiration is, in my opinion, essential. Although relaxants are widely used to obtain this I personally prefer deep ether with rapid manual closed-circuit overventilation. Whatever technique is used, provided the infant is pink and respirations controlled the result should be safe and satisfactory.

Technique of Repair

In our first 17 cases from 1948 to July 1953 we employed the right-sided extrapleural thoracic approach (Fig. 5). This approach is now only of historical interest in that it was used in the first successful case surviving the direct operation done by Haight[11] in 1943. For many years it proved a reasonable procedure, and some surgeons employed this approach as recently as September 1956.[20] Since July 1953, however, we have regularly employed a right-sided transpleural thoracotomy, either with resection of the 3rd or 4th rib or through the corresponding interspace. I believe this is quicker and gives better access to the anomaly and most authors agree with this opinion.[21]

On opening the pleura the lung is gently retracted downwards, the azygos vein divided, and the mediastinal pleura posterior to the trachea opened. Although the lower bulbous end of the upper blind pouch can, as a rule, readily be isolated and freed from the back of the trachea into the neck, it is preferable to isolate the upper fistulous end of the lower oesophagus first. Control of the fistula prevents further abdominal distension and makes ventilation easier. The oesophagus is isolated behind the right bronchus and as it is followed upwards it is carefully separated from the trachea. A small rubber catheter is passed around it for traction and the fistula isolated.

We have found great variation in the site of attachment of the fistula—once to the right main bronchus and twice at the level of the carina, but in the remainder always into the trachea but at a variable distance from the lower end of the pouch, several quite close to it—in a few 1 cm. distal but in most a little further down the trachea and in several 1 inch (2·5 cm.) beyond.

Once the fistula has been isolated an interrupted 00000 Deknatel minimal suture is passed through the upper border of its tracheal attachment. The fistulous track is now, divided, leaving a little cuff on the trachea as further interrupted sutures are placed and tied. The fistulous end of the oesophagus is now cut obliquely at a level where its lumen and musculature are adequate for anastomosis. A posterior layer of interrupted sutures are now passed from the pouch to the oesophagus (usually 6-8 sutures are possible) and *after* they have all been passed the tension is taken up on all of them and they are then tied individually. The pouch is now opened and a posterior interrupted mucosal layer is completed.

I now pass a fine catheter down the oesophagus and up through the pouch into the pharynx, from where it is partially withdrawn by the anaesthetist. The anterior mucosal layer of sutures is now inserted, with knots on the mucosa—the catheter prevents this layer from picking up the posterior mucosal layer. Finally the anterior muscular layer is placed; this is often difficult because the lower end is attenuated. The thicker pouch is then, as it were, rolled over on to the lower end and attached there. We have no hesitation, where necessary, in using only two layers of all-coats through-and-through sutures. The indwelling catheter is now withdrawn from the mouth. The anastomosis is anchored to the vertibral bodies to limit tension on the suture line. Penicillin (1 megaunit) and streptomycin G1 are placed around the anastomosis and a catheter is passed through the 9th intercostal space for underwater drainage and the incision is completely sutured

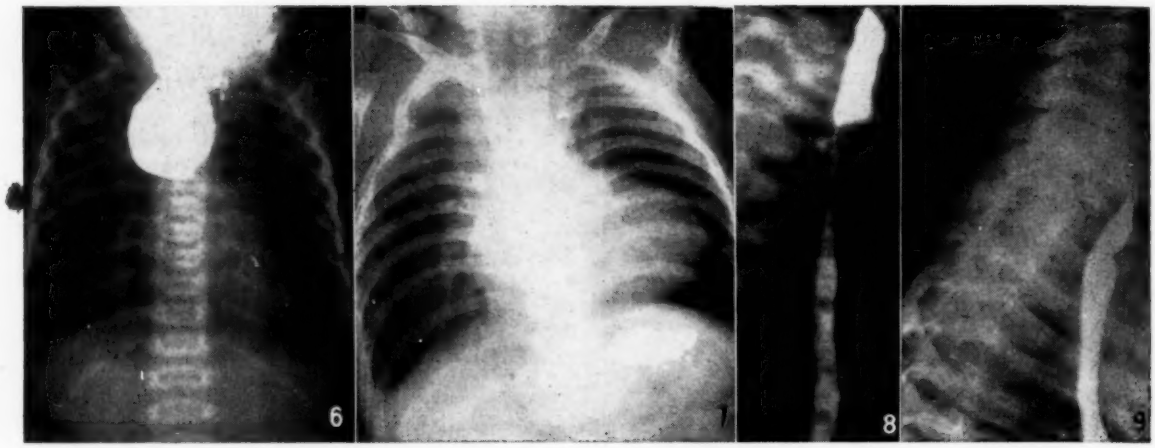

Fig. 6. Radiograph from an early case showing gross over-distension of pouch with lipiodol.
Fig. 7. Radiograph 10 days after successful surgery confirming patency of anastomosis in a case with the tetralogy of Fallot shown by the boot-shaped heart.
Fig. 8. Radiograph demonstrating local extravasation of lipiodol at site of anastomosis 5 days after surgery.
Fig. 9. Radiograph showing minimal narrowing at site of anastomosis two years after uneventful surgery in the first successful case.

in layers. If the lung has remained atelectatic, bronchoscopic toilet is again performed.

During the operation the blood loss is replaced and we have usually diluted 100 c.c. of blood with 100 c.c. of normal saline to ensure that there is no blockage of the polythene catheter during surgery. About 100-125 c.c. of this mixture is usually administered.

POST-OPERATIVE CARE

1. The infant is returned to the ward in an oxygen incubator which is humidified or into which alevaire is introduced.

2. The pre-operative antibiotics are continued with for 7-10 days. Broad-spectrum antibiotics are prescribed when indicated.

3. Cautious pharyngeal suction through a soft rubber catheter is employed when necessary; it is essential in the first few hours, because oedema of the anastomosis does not allow the child to swallow its saliva.

4. The infant is turned from side to side every few hours to prevent hypostatic congestion and atelectasis. In 9 cases atelectasis was considered to be the cause of death.

5. Ascorbic acid and vitamin-B complex are given in the drip in the first few days and subsequently by injection or by mouth.

6. Fluid requirements are most carefully estimated and continued by intravenous drip. Great care is taken not to overload these infants and in the first few days we only use 5% dextrose. On the 3rd day 50 c.c. of Darrow's solution is given, and on the 4th day 50 c.c. of infants' plasma or serum. Sodium chloride has not been given in our cases in view of the saline-diluted blood transfusion which is given during the operation and continued very judiciously in the first few post-operative hours as indicated by the child's general condition and loss through the underwater catheter.

7. A bedside portable X-ray of the chest is taken on the 1st post-operative morning to assess any contralateral atelectasis and ipsilateral haemothorax or pneumothorax.

8. The intercostal catheter is usually removed on the 2nd post-operative day.

9. On the 4th post-operative morning if the anastomosis has been a satisfactory one, or on the 5th day if the anastomosis has been a little under tension, one drachm of sterile water is given orally drop by drop to lubricate the pharynx, as it were, and to stimulate swallowing. Half a drachm of lipiodol is then given orally and a portable X-ray is taken after 2 minutes and again after 5 minutes—the latter to disclose any delayed leak at the anastomosis and to confirm that the lipiodol has entered the stomach (Fig. 8). In 3 cases (11, 17 and 20) disruption of the anastomosis was evident, and resulted in death. In case 21 there was considerable delay at the cardio-oesophageal junction, and as no relief was obtained with Eumydrin a feeding gastrostomy was performed by Mr. W. Kark on the 8th day after surgery. This child subsequently did well and the gastrostomy was closed 3 months later.

10. Once the lipiodol swallow has shown that the anastomosis is patent and intact, a careful pipette feed of 1 drachm of sterile water with added streptomycin is given slowly each hour. The following day 2 drachms of dextrose water with streptomycin are given by mouth and if the infant has been able to cope with this quite satisfactorily ½ drachm of expressed milk is given and the amounts gradually increased until the infant is taking a normal diet for its age and weight. If larger or quicker feeding is embarked upon the child avidly takes the feed and, owing either to the trauma of the operation or possibly to some lack of adequate innervation of the pharyngo-oesophageal mechanism, choking and inhalation tend to ensue.

11. If convalescence is straightforward we discharge the patient as soon as possible to avoid the risk of long-continued hospitalization. Four have been discharged on the 10th day.

12. Gastrostomy has *not* been made a routine in our cases. Gross,[22] from a large experience in Boston, strongly recommends it to take the strain off the anastomosis. I feel it is an added surgical procedure not devoid of risk and that early

feeding is the best method of dilating the oesophagus. With this latter view Potts[21] agrees.

POST-OPERATIVE COMPLICATIONS AND MORTALITY

1. *Immediate*

(*a*) Anaesthetic deaths, e.g. case 2, where spontaneous respirations did not return.

(*b*) Death from operative shock in the premature babes.

(*c*) Death due to inadvertent or erroneous pre-operative administration of barium, which caused uncontrollable bronchitis in 2 cases.

(*d*) Two cases died from associated anomalies, viz. ventricular septal defect and myelocele.

2. *Early*

(*a*) Respiratory complications, which are inherent in the anomaly since the tracheobronchial tree is assaulted both by aspiration of infected material from the blind proximal pouch and by reflux of gastric secretions through the fistula:

(i) Atelectasis—segmental (unimportant), lobar (usually transient) or pneumonic (usually fatal).

(ii) Bronchitis and bronchopneumonia.

(iii) Pulmonary oedema from intravenous overload.

(iv) Anoxia—in the premature and in those operated upon too early.

Nine cases died from pulmonary complications; 2 cases recovered from mild transient atelectasis, and 2 suffered repetitive attacks of inhalation pneumonitis from incoordination of swallowing and 1 from cardiospasm.

(*b*) Due to disruption of the anastomosis:

(i) Empyema with oesophago-pleural fistula: This occurred in 3 of our cases.

(ii) Oesophago-cutaneous fistula.

(iii) Recurrent tracheo-oesophageal fistula.

These complications are usually fatal.

(*c*) Digestive disturbances: Diarrhoea when oral feeding is commenced is not unusual and is probably due to the irritant effect of food on the sterile empty bowel. In case 25 it proved intractable and the child died of a second attack of diarrhoea 2 months after operation.

(*d*) Oedema: due to protein lack or, more usually, excessive administation of salt.

3. *Delayed*

(*a*) Lung abscess resulting from infection in an area of atelectasis or from inhalation of food in the early weeks of feeding before the swallowing mechanism has fully developed. This occurred in case 10, who has survived with a residual bronchiectatic abscess.

(*b*) Diarrhoea due to staphylococcal enteritis or moniliasis.

4. *Late*

(*a*) Recurrence of tracheo-oesophageal fistula.

(*b*) Stricture at site of anastomosis (Fig. 9). This occurred in 5 cases (3, 4, 10, 13 and 20) and resulted in multiple minor incidents of aspiration episodes and in retention of oesophageal foreign bodies, to which these children appear prone. All the strictures have responded to dilatation.

(*c*) Bronchiectasis from obstructive inhalation pneumonitis developed in 2 cases.

RECOVERY RATE

The recovery rate of infants operated on for congenital tracheo-oesophageal fistula as reported by various authors, and in our own series, is indicated in the following table:

Author	*Year*	*Patients*	*Recovery (No.)*	*Recovery %*
Cameron Haight[23] ..	1952	131	57	43·4
R. E. Gross[22] ..	1953	233	109	46·8
C. Koop, *et al.*[24] ..	1954	74	34	45·9
H. W. Clatworthy[25]	1955	35	17	48·6
R. Nicks[26]	1955	14	5	36
R. Shaw[27]	1955	61* 38†	25	41
Willes Potts *et al.*[21]	1957	113	—	54
D. I. Adler and D. N. Fuller	1949-56	34	17	50

* 1938-53. † 1948-53.

CONCLUSIONS AND SUMMARY

Congenital tracheo-oesophageal fistula with atresia is readily recognized clinically by excessive frothy oral mucus. Other congenital abnormalities are common but usually of a minor nature. Treatment cannot be delayed. Surgical repair offers a 50% survival rate and is possible only because of modern anaesthesia, fluid replacement and careful nursing.

The subject is reviewed, and the author's experience with 34 cases operated on at the Children's Hospital, Johannesburg, is reported.

The successes in this series have been attributable to the wholehearted cooperation and supervision of the paediatricians at the Transvaal Memorial Hospital for Children, to whom I would like to express my deep appreciation viz. Drs. Seymour Heymann, S. N. Javett, A. E. Strawbaun and Jacques Theron. Sister Mackay has been in charge of these patients and to her and her staff I would like to pay tribute for their unfailing interest and meticulous care in their management. To successive housemen the survivors owe their existence. Of the 34 cases, 11 were operated upon by Mr. Dennis Fuller and one by Mr. G. Katz.

REFERENCES

1. Vogt, E. C. A. (1929): Amer. J. Roentgenol, **22**, 463.
2. Imperatori, C. J. (1939): Arch. Otolaryng., **30**, 352.
3. Saunders, R. L. (1947): Canad. Med. Assoc. J., **57**, 237.
4. Franklin, R. H. (1948): Ann. Roy. Coll. Surg. Engl., **2**, 70.
5. Gibson, T. (1682): *Anatomy of Human Bodies epitomized*, p. 240. London.
6. Ladd, W. E. (1950): Pediatrics, **6**, 9.
7. Lanman, T. H. (1940): Arch. Surg., **41**, 1060.
8. Leven, N. L. (1941): J. Thorac. Surg., **10**, 648.
9. Richter, H. M. (1913): Surg. Gynec. Obstet., **17**, 397.
10. Shaw, R. (1939): J. Thorac. Surg., **9**, 213.
11. Haight, C. and Townsley, H. A. (1943): Surg. Gynec. Obstet., **76**, 672.
12. McGregor, A. L. (1949): S. Afr. Med. J., **23**, 317.
13. Roberts, F. W. (1950): S. Afr. Med. J., **24**, 167.
14. Franklin, R. H. (1947): Lancet, **2**, 243.
15. Singleton, A. and Knight, M. D. (1944): Ann. Surg., **119**, 556.
16. Belsey, R. H. (1949): Proc. Roy. Soc. Med., **42**, 915.
17. Haight, C. (1944): Ann. Surg., **120**, 622.
18. Franklin, R. H. (1949): Proc. Roy. Soc. Med., **42**, 507.
19. Belsey, R. H. and Donnison, C. P. (1950): Brit. Med. J., **2**, 324.
20. Humphries, G. H. *et al.* (1956): J. Thorac. Surg., **32**, 332.
21. Potts, W. and De Boer, A. (1957): Surg. Gynec. Obstet., **104**, 475.
22. Gross, R. (1953): *Surgery of Infancy and Childhood*. Philadelphia: Saunders.
23. Haight, C. (1952): Ann. Surg., **136**, 717.
24. Koop, C. E. *et al.* (1954): Surg. Gynec. Obstet., **98**, 687.
25. Clatworthy, H. W. (1958): Pediatrics, **16**, 122.
26. Nicks, R. (1955): N.Z. Med. J., **54**, 682.
27. Shaw, R. (1955): Ann. Surg., **142**, 204.

American College of Chest Physicians 1959 *Prize Essay Contest*. Three cash awards are offered in this contest (1st prize $500.00, 2nd prize $300.00, 3rd prize $200.00) which is open to *undergraduate* medical students throughout the world. Essays may be written on any phase of the diagnosis and treatment of chest diseases (cardiovascular or pulmonary). The contest closes on 15 April 1959 and further information may be obtained from: American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois, USA. In the 1958 contest Miss June Hagen of the University of Cape Town Medical School received an Honourable Mention for her essay on 'Cryptococcosis of the Lung'.

PYODERMA GANGRENOSUM COMPLICATING A SIMPLE SKIN GRAFT

H. KLEVANSKY, M.B., CH.B. (RAND), M.R.C.P. (EDIN.) and R. C. B. MACRAE, M.B., CH.B. (EDIN.), F.R.C.S. (ENG.), *Port Elizabeth*

A 43-year-old female, when first seen, had an ulcer on the outer side of the left leg about 3 inches above the level of the ankle joint. The ulcer was deep, extended down to the deep fascia and was approximately $3\frac{1}{2}$ inches long by $2\frac{3}{4}$ inches broad. The surrounding skin and subcutaneous tissues were thickened and inflamed, the edges and base of the ulcer were irregular, unhealthy looking and undermined. There were small blisters on 2 areas of the skin edge at the upper end of the ulcer.

The ulcer had been present for 5 weeks. It commenced with a very small irritable blister which the patient noticed shortly after rubbing an itchy spot which she assumed to be an insect bite. A ring of blisters formed around the orignal affected area; they soon burst and a scab formed. A week later, she says, she went down with 'blood poisoning', accompanied by a high temperature, aching limbs, enlarged and painful glands in the left groin, severe pain at the site of the blisters and much swelling of the ankle region. A deep ulcer now formed at the orignal infected site.

An injection of penicillin was given but, as it caused a severe reaction, it was not repeated. In spite of this reaction she took penicillin by mouth. The temperature subsided and the glands in the groin became reduced in size. The ulcer, however, remained extremely painful and continued to spread. The following dressings were applied: Ichthyol ointment, acriflavine emulsion, penicillin tulle, jelonet and furacin ointment. She says that whatever dressing was put on made the ulcer worse until furacin ointment was used. This dressing gave some relief but did not stop the ulcer from spreading.

Before the commencement of the ulceration the patient had been a very active, healthy and energetic woman, seldom ill, always busy with her home and children. In her spare time she helped in a store and shouldered the burdens and illnesses of her husband's employees and those of her neighbours. Her only departure from robust health had been septic infection of the tonsils on about 5 occasions in the previous 2 years. There was no apparent cause for the ulcer. Insect bites, such as that of a poisonous spider, were excluded on the history and absence of pain at the commencement. There was no sign or symptom suggesting a vascular cause.

The patient was admitted to hospital. A diphtheric infection was suggested as a possible cause and swabs were taken for culture and examination. The day following admission to hospital the tonsils became inflamed and a membrane formed on them lending strong support to the presumptive diagnosis. Swabs were taken from the throat. Direct examination and culture showed no diphtheria bacilli, but staphylococci and streptococci were found in both the ulcer and the throat.

Special Investigations

The following investigations, including excision of a portion of the edge of the ulcer for biopsy, were carried out with results as shown:

Throat swab (29 *June* 1957): Microscopic examination showed moderate mixed bacteria, mainly gram-positive cocci and scanty pus cells. Cultivation yielded a profuse mixed growth of non-haemolytic streptococci and *Staphylococcus aureus*.

Cultures from wound (2 *July* 1957): Microscopic examination of cultures from the new ulcerated area showed numerous pus cells and gram-positive cocci resembling staphylococci and scanty streptococci. Cultivation yielded a profuse growth of *Staphylococcus aureus* together with scanty non-haemolytic streptococci. Microscopic examination of cultures from the old ulcerated area showed numerous pus cells and scanty staphylococci. Cultivation yielded a profuse growth of *Staphylococcus aureus* together with scanty non-haemolytic streptococci.

Blood count (9 *July* 1957): Haemoglobin 82·5% (12·2 g.%), colour index 1·01, erythrocytes 4,000,000 per c.mm., leucocytes 12,250 per c.mm., neutrophils 55%, monocytes 1%, lymphocytes 34·5%, eosinophils 9·5%, basophils nil and PCV 35%. The red cells were normal in appearance. There was an eosinophilia.

Swabs (9 *July* 1957): The results of a number of sensitivity tests are given in Table I.

TABLE I. RESULTS OF SENSITIVITY TESTS

Antibiotics	*Staphylococcus aureus*	*B. proteus*	*Streptococci*
Erythromycin	++		—
Terramycin	+		++
Novobiocin	++	++	++
Chloromycetin	++	+	+
Aureomycin	+	—	++
Penicillin	—		—
Streptomycin		++	
Sulphatriad	—	+	+
Sulphamezathine ..		+	
Gantrisin		+	

Key: ++ Very sensitive. + Sensitive. — Resistant.

Blood count (25 *July* 1957): The following are the results of a subsequent blood count. Haemoglobin 88% (13·0 g.%), colour index 1·0, erythrocytes 4,400,000 per c.mm., leucocytes 11,800 per c.mm., neutrophils 50·5%, monocytes 4·5%, lymphocytes 33·5%, eosinophils 11·5%, basophils nil and PCV 37%. The red cells showed anisocytosis. There was an eosinophilia.

Pus (26 *July* 1957): Microscopic examination showed numerous pus cells and gram-positive cocci. Cultivation yielded a profuse growth of *Staphylococcus aureus*.

Blood (27 *July* 1957): The fragility of the red cells was within normal limits.

Culture from wound (31 *July* 1957): Cultivation for fungi was negative.

Faeces (2 *August* 1957): The ultraviolet light test for porphyrins gave a negative result on this specimen.

Diagnosis and Treatment

Several attempts were made to find and culture anaerobic organisms with negative result. Chronic infection, such as tuberculosis and syphilis, were considered and excluded on the short history, rapid progression of the ulcer and absence of physical signs of such diseases in other parts of the body. The W. R. was negative. Allergy was considered as a possible cause, though it was hard to see to what she could be allergic. There was no history of any other allergic manifestation in her past history. Her reaction to penicillin occurred only after the trouble had commenced. It was concluded that she had lost resistance for some reason, possibly connected with her recurrent tonsillar infection. She was kept in bed with the leg slightly elevated. Saline dressings were applied and as the ulcer, after a few days, began to show signs of healthy granulations, arrangements

were made for a skin-grafting operation as soon as local conditions appeared satisfactory.

Three weeks after her admission to hospital, a Thiersch skin-grafting operation was done, the donor area being the outer and anterior part of the thigh. No undue symptoms occurred for a week—then she began to complain of pain in the thigh. When the dressings were removed 2 days later, the donor area was seen to be heavily infected—the thigh around the area was swollen and inflamed, the temperature rose rapidly and once again the glands in the groin became enlarged and very tender. The graft appeared to have taken in part, though later it sloughed off completely. Two deep ulcers rapidly appeared on the donor area and spread down to the fascia lata of the thigh, and large sloughs of subcutaneous tissue formed under the intervening and surrounding skin.

The ulcer on the leg began to spread again and all the grafted skin sloughed off. Her condition became alarming, with general signs of a severe septicaemia developing. She was put on treatment with meticorten, 5 mg. *t.i.d.* Saline dressings were continued both on the original ulcer and on the new ulcerated area on the thigh. The response was satisfactory, but after 2 days, as she was still running an irregular temperature, novobiocin, 500 mg. was given in addition. A suggestion was now made that this was a case of pyoderma gangrenosum, due to agammaglobulinaemia or hypogammaglobulinaemia. A paper-electrophoretic test of blood proteins was at once carried out with the following result: Albumin fraction normal, alpha 2 globulin raised, Alpha 1 beta and gamma globulin normal. Estimation of blood proteins showed total protein 6·5 g. per 100 cc. albumin 4 g., glubulin 2·5 g. The only abnormality shown was the rise in the alpha 2 globulin, the significance of which is apparently unknown.

Under the influence of the new line of treatment the patient made rapid and satisfactory progress. The signs of septicaemia disappeared. The ulcers on the donor area filled in with remarkable rapidity once the sloughs came away. The original ulcer on the leg took on a healthy appearance with granulations, and comparatively slowly, but surely, began to heal from the edges towards the centre. Sixteen days after commencing the meticorten treatment the ulcers were so nearly healed and were making such good progress that she was able to return home.

She continued on the same treatment for 3 weeks. Her condition then became stationary, with a small unhealed area on both the leg and the thigh. It was then suggested that gamma globulin should be tried and accordingly injections of 20 cc. were given twice weekly for 2 weeks. There was no marked improvement locally, though the patient said she felt better after the injections. The position became static for the time being. It was then decided to try local cortisone treatment. She was put on terra-cortril ointment, and instructed to use only a very small quantity to cover the ulcers as thinly as possible. The result was dramatic and pleasing. She says that the pain was immediately relieved and that for the first time in 5 months she was able to sleep all night without any pain or discomfort.

This treatment was continued until the beginning of December, when the ulcers finally healed. When seen at the beginning of March this year, the patient had put on weight, felt well, was full of energy and enjoyed life once more. There had been no return of tonsillitis or other septic infections. The scars on the leg and thigh were sound though bluish in colour.

DISCUSSION

The remarkable ulceration which occurred in this patient spontaneously in the leg and followed a simple surgical operation to the thigh, and the failure to respond to antibiotics and conservative treatment, is characteristic of pyoderma gangrenosum (phagadena). The presence of spreading ulceration associated with pus and marked destruction of tissue is consistent with the diagnosis. The apparent inability of the patient to overcome infection and the recurrent attacks of purulent tonsillitis, suggest a low resistance to infection.

Pyoderma gangrenosum characteristically occurs in ulcerative colitis, but may occur without the latter. It may or may not be associated with an absence or lowering of the gamma globulins in the blood. The initial lesion may be an ulcer of the leg, which may extend, or new lesions may develop in other parts of the body, e.g. boils or furuncles, which rapidly break down and become gangrenous.

In our patient full blood counts did not reveal any evidence of any of the blood dyscrasias which may be associated with leg ulceration, e.g. leukaemia, sickle-cell anaemia, haemolytic jaundice, etc. General examination failed to reveal an enlarged spleen, liver or generalized adenopathy. Culture from the thigh and leg revealed staphylococci, on several occasions but on no occasion were anaerobic bacteria isolated. In spite of antibiotic therapy there was no evidence of improvement until prednisone was given. In order not to confuse the issue, when the latter was started all antibiotics were discontinued. There was a dramatic improvement in the appearance of both ulcers on prednisone therapy. An antibiotic, novobiocin, was only added later because of the possibility of spreading sepsis and the fact that we did not have the courage to continue without the cover of an antibiotic.

In view of the clinical picture and history of recurrent attacks of tonsillitis, the possibility of agammaglobulinaemia or hypogammaglobulinaemia was considered. A single blood examination revealed normal values. This is not considered a contraindication to gamma-globulin therapy by Sulzberger[1]. It was therefore decided to try injections of gamma globulin, and the patient was given 20 cc. weekly for 2 weeks. She stated that she felt much better, but there was no observable clinical improvement in the local condition of the ulcers. No inference can be drawn as the dosage was small in comparison to amounts which have been tried in similar cases.

SUMMARY

A case of pyoderma gangrenosum (phagadena), following a skin-grafting operation, is described. This patient showed a dramatic improvement only on prednisone therapy and was finally cured by the local use of hydrocortisone under cover of an antibiotic. Gamma-globulin therapy did not appear to have any beneficial effect on the ulcers.

REFERENCE

1. Sulzberger, E. (1957): Amer. J. Derm., 75, 917.

TREATMENT OF BILHARZIASIS WITH ANTIMONY DIMERCAPTOSUCCINATE

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W. O. HARRISON, *Mine Medical Officer, Havelock Asbestos Mine, Swaziland*

TWSb (antimony-a, a'-dimercaptosuccinate) was synthesized by E. A. H. Friedheim, New York. It is put up in powder form in multiple dose vials for intravenous (diluent 20-50% sterile hypertonic glucose solution) or intramuscular (diluent sterile normal saline) use. In these trials all patients were given the drug by the intramuscular route.

Material

The patients were all apparently healthy Bantu male recruits between 20 and 40 years old, from the Transvaal or Mozambique to Havelock Asbestos Mine in Swaziland. The mine is in a bilharzia-free area and the chances of reinfection were therefore minimal. Patients selected for treatment all had double infections with *Schistosoma haematobium* and *S. mansoni*.

Diagnosis was made by simple gravity sedimentation and examination of urine, and concentration of *S. mansoni* ova by sieving and sedimenting the faeces with water and pouring off the supernatant fluid until clear. One examination of urine and faeces was done. The cases were admitted to

TABLE I. TESTS OF CURE

Case	Dosage	Urine examinations		Faeces examinations		Rectal snip result	Side-reactions
		Number	Result	Number	Result weeks after treatment		
1	0·2 g.	10	Neg.	10	Neg.	Dead h. and m.	Nil
2	daily × 10	9	Neg.	2	Pos. (7)	Dead h. and m.	Nil
3		Not done		Not done		Not done	Nil
4		8	Neg.	9	Neg.	Not done	Nil
5	0·4 g.	Not done		Not done		Not done	Nil
6	daily × 5	9	Neg.	3	Pos. (8)	Dead h. and m.	Nil
7		10	Neg.	10	Pos. (10)	Dead h. and m.	Nil
8		Not done		Not done		Not done	Nil
9		4	Neg.	1	Neg.	Not done	Nil
10		3	Neg.	1	Pos. (10)	Dead h. Vi. m.	Nil
11		4	Neg.	1	Pos. (7)	Dead h. Vi. m.	Nil
12		2	Neg.	2	Neg.	Not done	Nil
13	0·34 g.	3	Neg.	2	Pos. (9)	Dead h. and m.	Nil
14		1	Neg.	1	Neg.	Not done	Nil
15	*t.d.s.*	4	Neg.	2	Pos. (6)	Neg	Nil
16		Not done		Not done		Not done	Nil
17	for 2 days	5	Neg.	4	Pos. (11)	Dead h. and m.	Coughing after 1st three injections
18		Not done		Not done		Not done	Vomiting after 4th injection, Temp. 100°F.
19		Not done		Not done		Not done	Vomiting after 4th injection
20		4	Neg.	1	Pos. (8)	Neg.	Vomiting after 4th injection, Temp. 99°F.
21		5	Neg.	5	Pos. (11)	Dead h. Vi. m.	Vomiting after 4th injection
22		5	Neg.	5	Neg.	Not done	Vomiting after treatment completed, Temp. 99°F.
23		8	Neg.	6	Pos. (11)	Dead h. and m.	Vomiting after treatment completed, Temp. 100°F.
24	0·4 g. 8-hourly × 3	Not done		Not done		Not done	Severe vomiting after third injection
25	0·4 g. 8-hourly × 5	3	Neg.	3	Neg.	Not done	Severe vomiting, abdominal pain, liver tenderness, bile in urine after 5th injection
26		3	Neg.	3	Pos. (10)	Not done	Nil
27		3	Neg.	1	Pos. (5)	Not done	Nil
28	0·4 g.	6	Neg.	5	Neg.	Not done	Same as No. 25 after 6th injection
29	8-hourly × 6	2	Neg.	2	Pos. (9)	Not done	Bile in urine after 6th injection
30		3	Neg.	2	Pos. (10)	Not done	Severe vomiting, bile in urine after 6th injection
31		1	Neg.	1	Pos.	Not done	Severe vomiting, bile in urine after 6th injection
32		3	Neg.	3	Neg.	Not done	Rise of temperature after treatment completed
33		3	Neg.	3	Neg.	Not done	Nil
34	0·4 g.	3	Neg.	3	Neg.	Not done	Nil
35	8-hourly × 7	2	Neg.	2	Neg.	Not done	Nil
36		5	Neg.	2	Pos. (4)	Not done	Nil

h. = haematobium m. = mansoni Vi. = viable

hospital for the duration of, and 1 day following, treatment.

Dosage Schedules

A. *Total dose* 2·0 *g.* (1) 0·2 g. daily for 10 days, 4 patients; (2) 0·4 g. daily for 5 days, 3 patients; and (3) 0·34 g. thrice daily for 2 days, 16 patients.

B. *Total dose* 2·4 *g.* 0·4 g. 8-hourly, 8 patients.

C. *Total dose* 2·8 *g.* 0·4 g. 8-hourly, 5 patients.

Schedule A was used initially until it was found that the curative effect on *S. mansoni* was poor, when schedules B and C were used.

RESULTS

Tests of Cure

Tests of cure were carried out from the 4th week onwards after completion of treatment. The total contents of the bladder was called for after hard exercise; it was sedimented by gravity, and the whole of the sediment examined for ova. Water concentration of the whole stool was used for the detection of *S. mansoni* ova. A rectal snip was done on some cases in addition to the examination of the faeces. Viability of ova was determined by producing motile miracidia from the ova. All positive cases recorded were discharging viable ova.

S. haematobium. None of the cases examined after treatment were found discharging viable ova of *S. haematobium.* This is in accordance with Alves' (1958) findings in Rhodesia.[1] He used a different dosage schedule (0·4 g. daily for 4 days) and a different (intravenous) route of administration.

S. mansoni. Of the 29 cases examined after treatment, 17 (56.6%) were still discharging viable *S. mansoni* ova and probably must be considered failures. *Table I* shows that there was no difference in the number of positives with the different dosages used. The superiority of faeces examination over 1 rectal snip is obvious; only 3 rectal snips contained viable ova in 11 cases with viable ova in their faeces. Several cases in whom *S. mansoni* ova were not found received a limited number of tests of cure. This was mainly due to their leaving the mine before sufficient tests could be done, and accounts for some cases not receiving any tests of cure

Side-reactions

Dosage schedules A (1) *and* (2). No side-reactions were noted in any of the 7 cases.

Schedule A (3). Of the 16 cases 9 had no side-reactions and 1 (No. 17) had coughing only after the first 3 injections. Six cases vomited; in 4 this happened after the 4th injection—2 had a concommitant rise of temperature, and the other 2 had a rise of temperature after the treatment had been completed. In the cases who vomited after the 4th injection, the last 2 injections were postponed for 8 hours. No further vomiting occurred and the temperatures subsided. In none of these 6 cases was the vomiting severe or prolonged.

Schedule B. Eight cases were put on this schedule. Two of them (Nos. 24 and 25) were unable to complete the course owing to severe side-reactions with signs of acute liver damage. One of these (No. 24) received a total of 1·2 g. TWSb in 24 hours and the other 2·0 g. in 40 hours. Of the remaining 6 cases 2 had no side-reactions, but 4 showed signs of liver damage, with bile in the urine, or severe and prolonged vomiting, or liver tenderness. These signs became evident after treatment had been completed and necessitated drastic action.

Schedule C. Strangely enough the only side-reaction in these 5 cases was a rise of temperature in 1 patient after treatment had been completed.

It was obvious that side-reactions in patients receiving a heavy dose of TWSb (0·4 g. 8-hourly) were severe and dangerous. With the less heavy doses (0·34 g. *t.d.s.*) side-reactions were too numerous, though in this series not dangerous, for the drug to be used in ambulant cases. With the light doses of 0·2 and 0·4 g. daily no side-reactions were noted, which supports Alves' findings in Rhodesia[1] on 25 ambulatory patients treated with 0·4 g. of the drug daily for 4 days.

Conclusions

1. TWSb is probably an effective drug in the treatment of *S. haematobium* in doses that do not give rise to severe side-reactions.

2. The drug is not effective in the treatment of *S. mansoni* in any dosage schedule used in the present series in patients from the Transvaal or Mozambique.

3. Side-reactions in patients with double infections of *S. haematobium* and *S. mansoni*, except those receiving light doses of the drug, were numerous, severe and sometimes dangerous.

SUMMARY

Thirty-six patients suffering from *S. haematobium* and *S. mansoni* infection were treated with TWSb.

Of the 29 patients who submitted themselves for re-examination after treatment none were found shedding viable ova of *S. haematobium*; 17 of them were found to be shedding viable *S. mansoni* ova.

Side-reactions were severe in patients receiving the larger doses of the drug.

We wish to thank Mr. L. A. West, Manager, Havelock Mine, for allowing us to do these trials, and other members of the mine staff for their cooperation; Dr. B. de Meillon, Honorary Director of Bilharzia Research, for his criticism, and the South African Council for Scientific and Industrial Research for permission to publish this paper.

1. Alves, W. (1958): Cent. Afr. J. Med., 4, 15.

TWO EXPERIMENTS IN AFRICAN WELFARE

Two experiments in African welfare, carried out by the Rhodesian mines of the Anglo American Corporation Group, have proved remarkably successful, Dr. J. H. G. van Blommestein, group medical consultant, discloses in his annual report just issued.

The first was the establishment of health centres in the villages where the African workers and their families lived. These centres had been functioning for 3 years and had proved themselves a necessary and indispensable adjunct to African family life, he states. The second was the training scheme introduced for Native female nurses. Two training schools had been established—at Wankie and Nchanga—and the response from Native women for training in these schools had been remarkable. It was now proposed to enlarge the school at Nchanga. In addition, feeding centres had also been established at which Native children of the lower age group were given fortified meals each day.

Dr. van Blommestein also states that malaria which, until recently, represented a major health problem among Native mine workers in Rhodesia, had been virtually eliminated by the use of residual insecticides. He warns, however, that only by constant maintenance of vigilance together with continued anti-mosquito control could the areas continue to enjoy the freedom from malaria which they did today.

Dr. van Blommestein also emphasizes the meticulous care which has to be devoted to the health of every individual Native employed on the mines. He states that large numbers of Natives are recruited from areas where life is extremely primitive and are thrust into an environment completely alien to them. Not only were their surroundings entirely different, but their whole mode of life was changed, and much time and effort, therefore, had to be expended in training them to meet the modern needs of mining practice; to inculcate into them the most simple rules of hygiene, while at the same time teaching them to become aware of the danger which could result from even minor injuries sustained in the course of their work.

All Native workers, before entering the mining industry, are required to undergo clinical, physical and X-ray examinations and are inoculated against enteric fever and tetanus and vaccinated against smallpox.

As a further prelude to starting work all new entrants to the industry are aptitude-tested, one of the chief purposes of which is to select potential Native supervisors, or boss boys, to serve as intermediaries between the European and Native workers. In certain circumstances Natives are put through an acclimatization procedure to accustom them to abnormal conditions they might encounter underground.

All through their mining career Native workers undergo periodic clinical and X-ray examinations to evaluate, constantly, the standard of their health. The X-ray examinations are in compliance with the regulations laid down by the South African Pneumoconiosis Act.

All the mine hospitals of the Anglo American Corporation are fully equipped to provide the most modern facilities for the treatment of both medical and surgical cases. Physiotherapy plays an important part in the post-operative treatment of injuries, while X-ray departments and laboratory facilities form an integral part of the hospital organization. In addition there are facilities for the treatment and rehabilitation of cases of industrial and non-industrial diseases and injuries.

Dr. van Blommestein, referring to the important part played by first aid, states that the proportion of Natives qualified in first aid is about 15 per cent of the total employed on most mines. In 1957, 2,000 Natives took part in first aid competitions in the Orange Free State mines. Not only was the knowledge they acquired most valuable in the mines but it became even greater when they returned to their homes where medical aid is not easily obtainable, as a rule.

UNIVERSITY NEWS

PROFESSOR OF ANATOMY, UNIVERSITY OF NATAL

Dr. E. N. Keen, who has been slected to succeed his father, Prof. J. A. Keen, as head of the Department of Anatomy at the University of Natal, obtained his medical training partly at King's College, London, and partly at the University of Cape Town, where he graduated M.B., Ch.B. in 1943. From 1944 to 1947 he was assistant government pathologist in Cape Town and lecturer in medical jurisprudence at the University of Cape Town. He was then for a period lecturer in anatomy, before going to Britain in 1949 for post-graduate study. He held a number of surgical posts in Manchester, and obtained the F.R.C.S. (Eng.) in 1951. In the following year he succeeded his father as senior lecturer in anatomy at the University of Cape Town, and has held this position until his new appointment. During this period he also obtained the M.D. degree of the University.

Dr. E. N. Keen

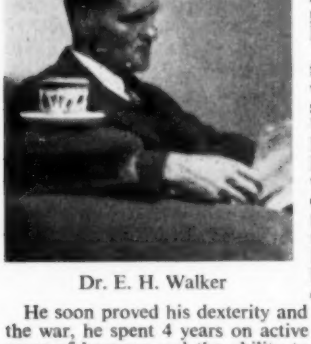
At Cape Town, Dr. Keen has established a reputation as a painstaking and stimulating teacher both of undergraduate and post-graduate students. As a research worker he has made notable contributions in the field of medico-legal anatomy and physical anthropology and also in that of the applied anatomy of the cardio-vascular system in man and animals.

As a member of the executive of the Lecturer's Association, Dr. Keen has taken an active part in University affairs, and he has been a valued member of several University clubs and societies.

IN MEMORIAM

DR. E. H. WALKER, M.B., CH.B., D.O.M.S., R.C.P.&S. (ENG.)

Mr. J. D. Joubert writes: Dr. Edward Holford Walker qualified in 1927 (M.B., Ch.B.) at the University of Cape Town, where he was a popular student and a good sportsman, particularly in boxing. After serving his house surgeoncies in Pretoria, he went to London to specialize as an ophthalmologist, which speciality he practised for a while in Pretoria.



Dr. E. H. Walker

During his active years he felt that he wanted more variety in his practice, and so he bought a practice in Umtata, and this is where Edward was at his happiest—he was a friend to all, he was welcome in every home, and every patient, no matter what race he belonged to, was treated in the same meticulous gentle way. He was a loyal colleague who would do anything for everybody.

He soon proved his dexterity and ability as a surgeon. During the war, he spent 4 years on active service. He had the keenest sense of humour and the ability to pick out the amusing things from every-day happenings. It still amuses me to think of an act he was persuaded to do often at parties, namely to demonstrate on a narrow couch how a Xosa woman disrobes her many layers of blankets before an abdominal examination, yet protecting her dignity.

He later went back to 'Eyes' and built up a large practice in Port Elizabeth. In the last couple of years he was not at all well, but he put up a valiant front. He died suddenly while fishing. He was perhaps one of the keenest fishermen I have ever known, and this, like everything else he did, was done in the right way and in the right spirit; in fact he made an art of fishing. One is glad for his sake that he should have enjoyed the last moments of his life at the sport he so loved.

To his wife Elsa, and the rest of his family, we can only say we are indeed sorry for the early departure of such a fine gentleman.

* * *

J. A. LEVITT, M.B., B.CH. (DUBL.), D.PHYS. MED.

Dr. H. D. Epstein, of Pretoria, writes: Dr. Jack Abraham Levitt passed away in Pretoria on Sunday 7 September after a prolonged illness. He was born at Petrusville in the Cape in 1899. In his early years Afrikaans was his home language and he developed a love for country life which he retained to the last. He received his medical education at the South African College, Cape Town, and then at Trinity College, Dublin, where he qualified in 1922. He married in Dublin, and on his return to South Africa, started practice at Middelburg, Transvaal, where he remained until the outbreak of war in 1939. At Middelburg he endeared himself to all by his humanity, capability and humility, and these char-

acteristics he retained to the end. He never spared himself and was available to help rich and poor alike at all times.

In the Army he was stationed at Voortrekkerhoogte Hospital and at first acted as general practitioner and later, after special study, as medical officer in charge of the physiotherapy department. In 1949, he obtained his Diploma in Physical Medicine at the Witwatersrand University. He practised in Pretoria as a specialist in physical medicine after his demobilization in 1946, and during that time was in charge of the department of physiotherapy at the General Hospital and the Meerhof Hospital.

Dr. Levitt knew the prognosis connected with his illness, which began 6 years ago, and as far as possible, gave still more of himself to his patients and those near and dear to him. In addition, he acknowledged the supreme power of the Ruler of the Universe and was greatly comforted by his beliefs.

As one who has known him intimately for the past sixteen years, I cannot speak too highly of his sincerity, his unselfishness and his kindness, and can truly state that he had many friends and no enemies. The medical profession in particular has been enriched by his life, and his family, friends and patients will miss him sorely.

* * *

DR. T. A. FULLER

Prof. E. C. Crichton, of Cape Town, writes: Tom Fuller was born in East London in 1890. His school days were spent at the South African College School, Cape Town. He studied medicine at Edinburgh University and qualified in 1913.

At Edinburgh he excelled in many branches of sport, particularly football and tennis, for which he was awarded his 'blue'. He joined the S.A.M.C. soon after the outbreak of the 1914-18 war and served in the ill-fated campaign at Gallipoli—and later in East Africa. He was mentioned in dispatches—a military cross would have been a more fitting award according to several men I have met, who served with him.

In 1922 he was appointed lecturer in the department of pathology and bacteriology at the University of Cape Town. He relinquished this post after about 2 years and joined Dr. Sutton Charnock in general practice. General practice did not satisfy him and he was never really happy in this sphere of medicine. Gradually he devoted more and more attention to anaesthetics and eventually proceeded to London in order to specialize.

In 1946 he was appointed head of the department of anaesthetics at Groote Schuur Hospital—he very soon realized that the best interests of the institution would be achieved by appointments on a full-time basis. With this object in view he reorganized the department and when he retired in 1952 he had the immense satisfaction of seeing a first class service in smooth working order.

He was a very good tennis player and won the Western Province championship. His handicap at golf, at one time, was plus 1. Nearly 2 years ago he became gravely ill as a result of a myocardial infarction and suffered considerable pain from attacks of angina pectoris until the time of his death.

He faced the inevitable with great courage and humour—never a word of self-pity escaped his lips as he saw the 'delectable mountains escape in the mist'.

He is survived by his second wife, a son and a married daughter.

* * *

DAMIAN HARMSEN PFEIFFER, M.B., CH.B., D.P.H.

Dr. P. G. J. Koornhof of Bloemfontein, writes: Dr. D. H. Pfeiffer died on 27 June 1958 at the age of 52 years. He had been Medical Officer of Health of Bloemfontein since October 1931.

Dr. Pfeiffer was born in Cape Town and was educated at the South African College School, where he obtained first-class passes in the Junior and Matriculation examinations. At the University of Cape Town he won a number of scholarships and bursaries and took the degree of Bachelor of Medicine and Bachelor of Surgery in 1928 and the Diploma of Public Health in 1931.

In 1929 he was appointed house surgeon at the Peninsula Maternity Hospital and house surgeon at the Somerset Hospital. In 1930 he held the position of senior house physician at the City Hospital for Infectious Diseases, Cape Town.

Dr. Pfeiffer will be remembered for his vast knowledge in a diversity of subjects, especially his knowledge of municipal regulations and public-health laws.

Our sympathy goes out to his bereaved wife.

* * *

JOHAN LOVIUS, M.B., CH.B., D.P.H.

Dr. P. G. J. Koornhof, of Bloemfontein, writes: Dr. J. Lovius died on 12 June 1958. He was 62 years of age. His death took place suddenly while he was on an overseas visit after retiring from the position of Deputy Medical Officer of Health of Bloemfontein last year. He died after a heart attack.

Johan Lovius was born in Bloemfontein in 1896 and received his early education at Brandfort. He attended Grey College, Bloemfontein, and then went to the South African College School, Cape Town. Thence he proceeded to Edinburgh University, where, in 1921, he took the degrees of Bachelor of Medicine and Bachelor of Surgery. He received his diploma of Public Health at the University of Cape Town in 1934. Dr. Lovius's studies at Edinburgh were interrupted when he joined the Navy during the First World War.

Soon after taking a public-health post in Bloemfontein, where he concentrated on the needs of the non-European population, he was appointed Deputy Medical Officer of Health.

Dr. Lovius was for many years District Commissioner at Bloemfontein of St. John's Ambulance Association and was decorated at Buckingham Palace and Government House for this work. He was also on the Executive Committee of the South African Cripple Care Association. He was a former President of the Jewish Guild.

Our sympathy goes out to his bereaved wife and family.

PASSING EVENTS : IN DIE VERBYGAAN

Mr. Isador Kaplan, F.R.C.S. (Eng.), F.R.C.S. (Edin.), has returned to Johannesburg after three years of postgraduate study in England and America.

* * *

Mr. G. P. Charlewood, F.R.C.S. (Edin.), F.R.C.O.G., has been invited by the Southern Rhodesian Medical Association to be the guest speaker at their congress to be held from 19-21 September 1958.

* * *

Mr. Basil de Saxe, F.R.C.S. (Edin.), general surgeon, has moved from Harley Chambers, Jeppe Street, Johannesburg to 1104 Medical Arts Building, Jeppe Street, Johannesburg. Telephone numbers remain unchanged.

* * *

S.A. Society of Anaesthetists, Cape Western Branch. The following office bearers were elected for 1958: Chairman: Dr. J. Abelsohn, Vice-chairman: Dr. S. Citron, Secretary/Treasurer/Area Representative: Dr. C. Harrison, Committee Members: Drs. J. Bam, G. Henderson and T. Voss.

Research Forum, University of Cape Town. The next meeting of research forum will be held at noon on Wednesday 1 October 1958 in the large A-floor lecture theatre, Groote Schuur Hospital, Cape Town. Dr. G. Selzer will speak on 'Poliomyelitis Viruses, Adaption and Combination'. All interested are invited to attend.

* * *

Mr. H. Gaylis, Ch.M., F.R.C.S., of Johannesburg has recently returned to Johannesburg having worked for a year in the Vascular Service of the Massachusetts General Hospital and Harvard Medical School, Boston, USA. At the annual meeting of The Society for Vascular Surgery in San Francisco, he delivered a paper on 'Arterial autografts'.

* * *

S.A. Orthopaedic Association, Cape Town Branch. Dr. J. E. M. Thomson a distinguished orthopaedic surgeon from Lincoln, Nebraska, will address a meeting of the Group in the E-Floor Lecture Theatre, Groote Schuur Hospital, Cape Town, on Thursday 2 October at 8 p.m. The subject of the address will be 'Rehabilitation of the Severely Handicapped'. All members of the profession are cordially invited to attend.

Mr. E. Abro, Ch.M., Urologist, wishes to inform his colleagues that in addition to his rooms at 704 Medical Centre, Jeppe Street, he has opened rooms at 102 Southern Medical Centre, Rosettenville, Johannesburg. Telephone: 26-2650.

Dr. E. Abro, Ch.M., Uroloog, wens sy kollegas in kennis te stel dat hy bykomstige spreekkamers te Southern Medical Centre 102, Johannesburg, geopen het. Telefoon: 26-2650.

* * *

Red Cross War Memorial Children's Hospital, Rondebosch, Cape. The next meeting in the Postgraduate Seminar Series of lectures held under the auspices of the University of Cape Town, Department of Child Health, will be held in the lecture theatre of this Hospital on Wednesday 1 October at 5 p.m. Dr. P. M. Smythe will speak on 'The Treatment of Tetanus Neonatorum with Intermittent Positive Pressure Respiration'. All medical practitioners are welcome.

* * *

A Scientific Meeting of the Association of Physicians of South Africa will be held in Cape Town on 8, 9, and 10 January 1959. Papers will be presented and part of the meeting will be devoted to a symposium on 'Pulmonary function testing'. It is hoped that as many members of the Association of Physicians as possible will attend. A congress dinner and a luncheon will be held. Physicians who are anxious to attend, are asked to communicate with Dr. H. Muller, Hon. Secretary, 812, Medical Centre, Heerengracht, Cape Town.

* * *

Supplementary Health Services. A symposium 'The Doctor and his Allies' by leaders of various supplementary health services: physiotherapy, speech therapy, occupational therapy, clinical psychology and radiography, will be held in the Harveian Theatre, Medical School, Johannesburg, on Thursday 2 October at 8 p.m. Prof. E. H. Cluver, Chairman of the statutory Supplementary Health Services Committee of the S.A. Medical and Dental Council and Chairman of the Council of Allied Medical Professions, will preside and the theme will be that modern medical practice is team work, the medical practitioner being increasingly dependent on the assistance of auxiliary groups. The opening talk will be by Dr. P. B. Peacock, Medical Officer of Health, Germiston. There will be 5 other speakers; demonstrations and 2 short films will be shown. The President will then sum up. A collection will be taken for the Dart Anthropological Institute Fund. It is hoped that the symposium will be attended by a large number of doctors so that they may be reminded of the valuable assistance that is becoming increasingly available to them for the implementation of modern medical science.

* * *

Cardiovascular Conditions. Scientific Conference at Kampala, Uganda, 14-16 January 1959. The East African Medical Research Scientific Advisory Committee extend a cordial invitation to anyone interested to attend the conference or take part in the proceedings. For the information of those wishing to present papers at the conference it is of importance that the Council be notified as soon as possible, and that notice should be given of the subject of the paper to be read, not later than 15 October 1958. It would also be appreciated if a summary of the paper (about 500 words) could be sent to the Council not later than the end of November, for incorporation in the conference programme. The paper should require about 20 minutes to deliver. It is proposed that about 16-18 papers shall be read during the 3-day conference. It is hoped that it may be possible to hold the conference in the Lecture Hall of the Makerere College Medical School, Kampala, but the venue and times for the meetings will be announced later. Further information may be obtained from: The Medical Research Secretary (A. T. Howell), Office of the East African Council for Medical Research, P.O. Box 30005, Nairobi.

* * *

World Medicine—Key to Peace. While the diplomats at the United Nations go through their laboured routines of negotiation, threats and compromise, in an effort somehow to keep the peace, it is a relief to be able to call attention to another kind of international meeting which recently occurred in Copenhagen—a meeting which held no possible foreboding of disaster, but promised nothing but good for all mankind. We refer to the twelfth general assembly of The World Medical Association, which brought together physician-delegates from 50-odd free nations for a benevolent exchange of scientific knowledge.

'When men search for peace through the processes of international politics, there is always implicit a frightful alternative, the possibility of war and death', declared an American physician, Dr. John Henderson, in the keynote address at Copenhagen. 'We physicians face no such dilemma', he continued, 'since we pursue only the objectives of the alleviation of suffering and the preservation of life'.

Dr. Henderson, who is medical director of Johnson and Johnson, New Brunswick, New Jersey, hailed the international exhibition of medical films, a feature this year for the first time of the WMA meeting, as a significant symbol of the fact that science knows no boundaries and speaks with a universal tongue.

While the life span in Western Europe and the United States has increased amazingly in recent years, Dr. Henderson said, age-old afflictions are still a major problem in the underdeveloped areas of the world. Disease breeds discontent and discontent breeds trouble.

'Through the co-operation of physicians and the total health team in all countries of the world', Dr. Henderson declared, 'there can be created a level of world health and world unity which could not otherwise be attained. This is the challenge which we of the World Medical Association acknowledge and accept.'

* * *

Union of South Africa, Department of Health. Newsletter No. 37 *of* 1958. Notification of formidable epidemic diseases and poliomyelitis in the Union during the period 5-11 September.

Plague and Smallpox. Nil.

Typhus fever: Cape Province. 13 (Thirteen) Native cases reported from Queenstown district. (Nine cases from the farm Bonaparte and four cases from the farm Narrow Point.)

Poliomyelitis

	Eur.	*Nat.*	*Col.*	*As.*	*Total*
Transvaal	3	2	–	–	5
Cape Province ..	2	–	2	–	4
Orange Free State	–	1	–	–	1
Natal	–	2	–	–	2
Totals	5	5	2	–	12

Correction:

Cape Province: 1 (One) European case reported in Newsletter No. 34 of 1958, rediagnosed as 'not poliomyelitis'.

Local Authorities		*Eur.*	*Non-Eur.*
Transvaal:			
Brakpan municipality	U		1
Delmas district	R	1	
Klerksdorp district	R		1
Krugersdorp district	R	1	
Pretoria municipality	U	1	
Cape Province:			
Cape divisional council	U		1
Uitenhage municipality	U	1	
Worcester divisional council ..	R		1
Kuruman divisional council ..	R	1	
Orange Free State:			
Thaba Nchu district	R		1
Natal:			
Bulwer district	R		1
Nkandla district	R		1

Correction:

Cape Province: 1 (One) European case from Kenhardt divisional council, reported in annexure to Newsletter No. 34 of 1958, rediagnosed as 'not poliomyelitis'.

R = Rural. U = Urban.

* * *

Die Tweede Akademiese Jaardag van die Geneeskundige Fakulteit van die Universiteit van Stellenbosch en die geneeskundige personeel van die Karl Bremer-Hospitaal, Bellville, is op Vrydag 5 September 1958 in die Burgersentrum, Bellville gehou. Besonderhede van die geleentheid is soos volg.

1. 'n Aantal voorlesings en referate is gelewer deur lede van die

doserende personeel. Al die referate was van 'n hoë gehalte en het getuig van verantwoordelike navorsingswer. 'n Prys vir die beste voordrag is toegeken aan dr. J. C. de Villiers van die Departement van Anatomie vir sy besondere referaat oor Kongenitale polikisteuse niere. Dr. de Villiers het reeds jare gelede as algemene praktisyn op Swellendam met hierdie navorsing begin. As beoordelaars het opgetree prof. J. F. du T. van Zyl, Dekaan van die Geneeskundige Fakulteit, prof. F. Forman van die Universiteit van Kaapstad, en dr. G. M. Malan, President van die Tak Wes-Kaapland van die Mediese Vereniging.

2. 'n Aantal wetenskaplike uitstallings is vertoon wat aspekte van die kliniese- en navorsingswerk aan die mediese skool illustreer. Ook hier is 'n prys toegeken vir die beste uitstalling en dit is toegesê aan dr. J. J. W. van Zyl vir sy film oor die Tegniek van binneaarse infusies.

3. 'n Groot en interessante uitstalling van farmaseutiese produkte en instrumente is gereël deur al die bekendste farmaseutiese firmas in die land. Dit dien ook gemeld te word dat hierdie firmas, deur hul finansiële steun, die uitvoering van die program van dié jaardag moontlik gemaak het.

4. Die verrigtinge van die dag is afgesluit met 'n dinee-dansbyeenkoms. By hierdie geleentheid was die Administrateur van Kaapland, Dr. J. H. O. du Plessis, as eregas en spreker aanwesig. Die Administrateur het by vernuwing die belangstelling en steun van sy Administrasie toegesê aan die twee mediese skole van Kaapland. Met betrekking tot die toekomstige opleidingshospitaal van die mediese skool van Stellenbosch, het hy gesê dat die Administrasie dit in die vooruitsig stel om 'n groot, nuwe, moderne hospitaal met 1,000 beddens te Parow, vir hierdie doel te bou. Die Dekaan van die fakulteit, prof. van Zyl, het in sy antwoord daarop gewys dat die toekomstige beleid insake die bou van nuwe hospitale rekening moet hou, nie net met die groot koste van hospitale nie, maar ook met sulke belangrike vraagstukke soos dié van die voorsiening van verplegingspersoneel. 'n Nuwe beleid sal ontwerp moet word wat alle aspekte van hospitaaladministrasie omsluit.

REVIEWS OF BOOKS : BOEKRESENSIES

BURNS

Burns—Pathology and Therapeutic Applications. By Simon Sevitt, M.D., M.Sc., M.A., F.R.C.P.I., D.P.H. Pp. xii+364. 85 Figures. 43s. 3d. + 1s. 7d. Postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1957.

This book is the first comprehensive volume to be devoted to the subject of burns together with the pathology and therapeutics thereof. When virtually all that is known about the subject is correlated and presented by one with such a wealth of personal experience as has Dr. Sevitt, it becomes necessary to study the contents in detail.

The subject matter is logically arranged. The author traces what happens in the burned area, the effects of hyperthermia on epithelial and other cells, and the acute inflammatory changes that follow. Excellent chapters are those on the role of infection in burns, on the epidemiology of infection, and on the control of the organisms causing infection.

Of inestimable value to the clinician are the chapters on burn shock, which has such severe effects on the circulation and on the distribution of body fluids and electrolytes. One is left in no doubt about the extreme urgency of the adequate plasma and electrolyte therapy that is necessary if burned patients are to be resuscitated in the early stages.

The effects of burning on the internal organs and on hormonal mechanisms are accorded due prominence, and the closing chapters are devoted to a brief, but interesting and instructive resumé of what is known concerning electrical, chemical and atomic burns, and the effects of radiation.

The basic trend of experimental and histopathological discussion is nicely offset by recommendations in regard to therapy in its widest sense, including the judicious and timely application of surgical measures. The author has brilliantly succeeded in his aim of 'letting all schools of thought contend', but equally has not shirked from giving his own opinions which are highly practical and extremely valuable.

This work is yet another fine contribution from one of the team at the Birmingham Accident Hospital, who have done so much to advance our knowledge of burns. Effective treatment of the burned patient is essentially a matter of team-work and in this volume every member of the team will find ample food for thought; and sound advice for application in his own special field.

D.McK.

ELECTROTHERAPY AND ACTINOTHERAPY

Clayton's Electrotherapy and Actinotherapy. A Text-book for Student Physiotherapists. 3rd Edition. By Pauline M. Scott, M.C.S.P., T.E.T., T.M.M.G. Pp. x+427. 207 Figures. 27s. 6d. London: Baillière, Tindall and Cox Ltd. 1958.

The 3rd edition of this well-known text-book for student physiotherapists has been revised to meet the requirements of the electrotherapy section of the syllabus of the Chartered Society of Physiotherapy. The book provides an excellent account of the physical principles underlying electrotherapy and actinotherapy, and is therefore a useful reference book for practising physiotherapists, as well as for members of the medical profession who use electrical apparatus. It is regretable, however, that no reference is made to ultrasound, due perhaps to the fact that this subject does not appear in the syllabus of The Chartered Society of Physiotherapy. The new chapters contain an account of the important advances in the production of valve generated muscle stimulating currents opening up the fascinating field of exponential currents. A short chapter on long wave diathermy provides an anachronism in a modern book, but is of historic and academic interest.

A.R.

THE GUINEA PIG IN RESEARCH

The Guinea Pig in Research. Biology—Nutrition—Physiology. By Mary Elizabeth Reid, Ph.D. Pp. 87. 7 Figures. Washington: Human Factors Research Bureau, Inc. 1958.

The term 'guinea pig' has long been used to signify a living being which is experimented upon. It is indicative of how much the gentle cavy has been used for this purpose and of how valuable it has been. Dr. Reid's publication is a mixture of review article and testimonial, but nevertheless contains a good deal of useful information.

Practically the whole range of biological research in which the guinea pig has been used, is reviewed and a fairly good selection of reference material cited. The 227 listed references are, of course, not not exhaustive of the literature on guinea pigs. Dr. Reid has been necessarily selective, but one feels that some important reference material has been omitted. No reference, for example, has been made to the contributions made by H. M. Bruce and A. S. Parkes to the knowledge of guinea pig nutrition, growth, and breeding.

This small volume should prove to be of value to anyone interested in the physiology of the guinea pig and the uses of this animal in biological research.

A.G.R.

SURGICAL MANAGEMENT

Principles of Surgical Management. By H. A. F. Dudley, F.R.C.S.E. With the Assistance of B. C. Paton, M.R.C.P.E., I. F. McLaren, F.R.C.S.E., I. B. Tait, F.R.C.S.E. and G. F. Murnaghan, F.R.C.S., F.R.C.S.E. Pp. viii + 203. 68 Figures. 27s. 6d. net + 1s. 9d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

Every now and then a medical book appears which one feels really needed to be written. It would be hard to equal Ham's *Histology*—perhaps the best text-book in the English language for clarity and comprehensiveness in a basic science; but amid a welter of publications on pre- and post-operative care *Principles of Surgical Management* stands out like a beacon.

The modern surgeon must have a physiological background. This much is common cause, but there is no doubt that the dog-

surgeons have inflicted a vast amount of tedious writing on long-suffering readers of surgical literature. Thus, they have terrified the older surgeon out of his wits with their disquisitions on intravenous therapy so that he has come to regard their milli-equivalents with almost superstitious awe. There is much more in surgical care than chemical juggling and it is the virtue of this book that this is recognized. For the trainee and for the older surgeon, who wants to brush up painlessly on the surgical facts of life, this product of the Edinburgh school is to be recommended. Professor John Bruce in a foreword writes: 'I have enjoyed reading this little book, indeed I have profited from the opportunity and I commend it unreservedly'. The present reviewer heartily concurs in these sentiments.

The authors have been fortunate in their choice of publishers. The paper and printing and illustrations are quite outstanding.

G.S.

CORRESPONDENCE : BRIEWERUBRIEK

LONG-ACTING PRESENTATION OF DRUGS

To the Editor: You conclude your Editorial[1] in the *Journal* of 23 August with the following paragraph: 'Probably methods such as these of presenting drugs in long-acting form, might with advantage be extended to other drugs that are administered by mouth.'

I would like to point out that for more than a year Belladenal retard has been available in South Africa, and that on numerous occasions advertisements on this preparation have appeared in your *Journal*. Belladenal retard is a new form of the well-known preparation 'Belladenal', and its mode of action is similar to the one described in your editorial. Belladenal retard combines the total belladonna alkaloids and phenobarbitone and by using different coatings, the release of the active ingredients is spread over a period of approximately 8 hours, so that the ingestion of 1 tablet Belladenal retard gives effective anticholinergic and sedative action over a period of approximately 10-12 hours. Belladenal retard is particularly indicated for the treatment of peptic ulcer and has the great advantage of affording throughout the night effective anticholinergic activity with one evening dose, thus reducing gastric secretion and motility throughout the night.

You will probably also be interested to hear that shortly Bellergal retard tablets will be introduced in this country, a preparation which is built on the same principle as Belladenal retard and will make available to the medical profession a long-acting form of the popular Bellergal used for the treatment of many psychosomatic conditions.

Paul G. Stein

Sandoz Pharmaceutical Department
P.O. Box 4461
Johannesburg
17 September 1958

1. Editorial (1958): S. Afr. Med. J., 32, 844.

ACUTE APPENDICITIS IN AN INFANT

To the Editor: Dr. Theunis Coetzee's account of acute appendicitis in a 9-days-old infant in the *Journal* of 6 September 1958[1] recalls a similar case that I saw some years ago at Crumpsall Hospital, Manchester. This infant was born in the hospital and was entirely breast-fed. He became ill on the 7th or 8th day, but there were no localizing signs. Apart from restlessness and a raised temperature, the main feature, as Dr. Coetzee observed in his case, was an increasing distension of the abdomen.

At the post-mortem examination gangrenous appendicitis with generalized peritonitis was found. In discussing this case at the time with other members of the staff, it was agreed that the lack of localizing signs made a correct diagnosis of acute appendicitis in a newborn infant extremely difficult.

G. Maizels

Martian House
Strand Street, Port Elizabeth
12 September 1958

1. Coetzee, T. (1958): S. Afr. Med. J., 32, 890.

RADIATION HAZARDS

To the Editor: May I crave the courtesy of your columns to be allowed to ask Mr. Franklin Bishop[1] a few questions on the subject of his letter on 'radiation hazards'.

He states: 'furthermore, Dr. Weinbren seems to think I have never seen radiation damage following X-rays. This I can assure you is not so. I have not only seen diagnostic radiation damage, but have actually seen carcinoma develop in these diagnostic radiation burns.'

I feel it would be in the interest of the general public who have to undergo these examinations, the medical profession who order them and the diagnostic radiologists who carry them out, if he would please be more specific. Perhaps he would tell us how many such cases he has encountered? When, what sort and what number of diagnostic X-ray investigations preceded such disastrous results? Also, and this is most important, were these 'burns' induced by screening or radiography? And were they under the control of a qualified radiologist?

Finally, his statement: 'of course the problem of over-radiation is becoming an increasing problem in spite of what Dr. Weinbren says.' Such a broad generalization requires amplification. It may be valid when applied to the products of nuclear explosions, but it is certainly not proved in regard to diagnostic radiology and such *ex cathedra* utterances are apt to be more harmful than helpful and certainly unreasonably alarming to the public.

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17 September 1958

1. Bishop, F. (1958): S. Afr. Med. J., 32, 928.

APLASTIC ANAEMIA FROM PHOTOGRAPHIC CHEMICALS

To the Editor: In answer to an opinion regarding whether any of the chemicals in use by amateur and professional photographers could be a causative agent in aplastic anaemia, I quote hereunder the text of a letter from Dr. David W. Fassett, Director of Laboratory of Industrial Medicine at Kodak works, Rochester, N.Y.

'There are no recorded cases of aplastic anaemia as far as we know in either amateur or professionals handling black and white developers such as elon (Metol) and hydroquinone. We know of no such cases being encountered in the handling of other more complex developers such as colour developers.

In addition to this lack of evidence of aplastic anaemia in ordinary use, we have specific observations on many hundreds of individuals in our own plants, some of whom have been in contact with high concentrations of elon and hydroquinone throughout their lifetimes without any evidence of difficulty. This is true both for workers who are engaged in manufacturing or packaging these developing agents and also for workers in various laboratories who are engaged in continuous processing operations.

There are, in addition, several theoretical reasons which would make it unlikely that any difficulty of this sort would be noticed. In the first place these agents do not penetrate the skin readily, since they are non-volatile and there is no way of introducing them into the body under ordinary conditions of use. In the second place, experimental work with animals does not indicate that even very large doses of these materials cause any generalized effect on the bone marrow.'

One must mention, however, that allergic reactions due to many of the photographic chemicals of the phenylene diamene group, commonly known as 'Metol poisoning' are well-known entities. One might almost generalize in saying that any developing agent which stains the hands is likely to be a powerful allergen.

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10 September 1958

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